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Biofeedback and the TMJ Syndrome: An Electromyographic Study

by

Ⓒ Roger O. Gervais

A THESIS
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Supervisor
....
....
..L Norman Thomas..

Date..18..June..1984.......

Dedication

This work is dedicated to my wife Marie and our sons Aaron and Roshan who have patiently subsisted on rumours of a husband and father for far too long.

"There is no condition which cannot be ennobled either by a deed or by suffering." Goethe

Abstract

Masticatory muscle hyperactivity has been implicated in the onset and maintenance of the temporomandibular joint (TMJ) pain-dysfunction syndrome. Various relaxation therapies have been used in managing this condition, and biofeedback has emerged as one of the most promising. The present study was conducted with a view toward clarifying the differences in masticatory muscle activity or hyperactivity between a sample of subjects manifesting signs and symptoms of TMJ dysfunction and an equal number of clinically asymptomatic subjects.

Membership in the two experimental groups was determined on the basis of a self-report screening questionnaire and a clinical examination of potential TMJ subjects. A total of 48 female subjects between the ages of 18 and 49 (24 TMJ, 24 ASYMPTOMATIC) participated in a nine-step stress experiment. The experimental procedure consisted of a baseline condition followed by four stressors each succeeded by a recovery period of equivalent duration. The purpose of the experiment was to assess each group's differential reactivity to the various types of stressors as indicated by masseter and temporalis EMG activity monitored throughout the nine conditions.

A Hotelling's t^2-test was conducted on the outcome to test for significant differences in muscle tension between the two groups for each experimental condition. No significant differences ($p > .05$) in mean EMG levels were found between the TMJ and ASYMPTOMATIC groups.

A second Hotelling's t^2-test was performed on the mean range of EMG activity recorded for each group. As in the first analysis, there were no statistically significant differences ($p > .05$) between the TMJ and ASYMPTOMATIC groups for this measure.

Two further analyses examined the correlation between EMG levels and TMJ noises (clicking, popping, grating, etc.); and the palpation tenderness of the masticatory muscles in the TMJ subjects. No significant correlations were found found between EMG activity and joint sounds, but there was a significant positive correlation between muscle activity and palpation tenderness.

Although the differences between the two groups were not statistically significant, it was concluded that biofeedback is a potentially effective therapeutic modality in treating the TMJ syndrome patient presenting with muscle tenderness and hyperactivity. Future research should attempt to reduce the large within groups variance observed in the present study by implementing more stringent screening and physical assessment procedures in assigning membership to the experimental groups. Finally, both dynamic and static measures of masticatory muscle activity should be included in further EMG studies of TMJ dysfunction.

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I. INTRODUCTION

Chronic pain related to dysfunction of the temporomandibular apparatus has, in the last thirty years, been the subject of intensive research within the dental community. Recently, however, the interest has spread into the broader medical and psychological professions leading to a growing acknowledgement of the temporomandibular (TMJ) pain-dysfunction syndrome. The syndrome which includes such symptoms as clicking and popping noises in the jaw joint, limitation of jaw opening, clenching and grinding of teeth, head, neck, and shoulder pain; subjective sounds in the ears, clogging of ears, and dizziness; low back and leg pain; hormone imbalances, chronic skin disturbances, respiratory ailments, and inflammation of the eyes; as well as gastrointestinal and gynecological difficulties, chronic fatique, poor stress tolerance, and depression, is seen by many progressive practitioners as a key to understanding and treating the phenomenon of chronic pain (Gelb, 1980). The TMJ syndrome involves the whole body; consequently, a complete view of the patient from both the physical and psychological dimensions is required. Current treatment of the TMJ syndrome favors a multi-disciplinary approach (Gelb, 1977, 1980; Morgan, House, Hall, & Vamvas, 1982).

In the last decade, biofeedback relaxation training was proposed as a possible treatment in the TMJ pain-dysfunction syndrome and since 1972 a number of studies have been published which report the success of this type of therapy.

The theoretical basis for the use of biofeedback training in the syndrome is the clinical observation that many TMJ pain-dysfunction patients manifest signs of excessive tension in the muscles of mastication, tension which appears to be related to stress and is directly implicated in some or all of the pain experienced. Biofeedback therapy is seen as a valuable means of directly promoting self-awareness and self-regulation of muscle tension in the patient. At this point, there are two presuppositions behind the use of biofeedback therapy in the TMJ syndrome. The first concerns the presence of excessive muscle activity in afflicted patients; the second involves the role of stress as a precipitating factor in the excessive muscle activity.

To date a number of studies have assessed the differences between TMJ and non-TMJ subjects with regard to experimental stress (Perry, Lammie, Main, & Teuscher, 1960; Yemm, 1968, 1969a, 1969b, 1969c, 1971). However, these studies used small sample sizes and did not establish very stringent control procedures for external variables. As a result, their conclusions cannot be reliably generalized. On the first point, there appears to be a complete lack of research into comparative muscle tension levels between normal and TMJ subjects, although the literature suggests that there is a difference between the two populations. Evidently, further research into normative differences in muscle tension and stress response patterns in TMJ patients and normal subjects is required to render the application of biofeedback therapy in the syndrome more precise and effective.

A. Overview

The present study used a nine (conditions) by two (groups) repeated measures across conditions format. Each of the nine experimental conditions used in the present study was designed to obtain electromyographic (EMG) data on all subjects for a resting baseline, the amount of deviation from baseline following presentation of the stressor, and post-stressor recovery. The EMG recordings were taken from the right and left temporalis and masseter muscles. On the basis of a self-report symptom questionnaire the subjects were assigned to a ASYMPTOMATIC or a TMJ group. Membership in the TMJ group was confirmed by a short clinical examination carried out by an experienced physiotherapist. Within two to six weeks following participation in the experiment, all but five of the TMJ subjects were given a complete dental examination by an orthodontist during which clinical signs of mandibular dysfunction were noted. Analyses of variance were conducted to test for significant differences in EMG levels between the two experimental groups, both in terms of individual muscle activity and summed activity for each experimental condition. Pearson product correlations were also obtained for the relationship between the number of questionnaire responses and EMG activity, and finally, between clinically observed signs of dysfunction and muscle activity.

B. Purpose

The principal hypothesis being tested in this study is that a sample of TMJ dysfunction subjects will differ significantly from a sample of normal, non-TMJ dysfunction subjects in terms of EMG activity at baseline, and following exposure to various experimental stressors. More precisely, it is expected that the TMJ group will demonstrate significantly higher EMG levels throughout the experiment as compared to the ASYMPTOMATIC group. Also, significant positive correlations are expected between the number of positive self-reported symptoms and clinical signs of dysfunction and the magnitude of recorded EMG activity. By clarifying the differences in muscle tension and response to stress between TMJ and ASYMPTOMATIC subjects, it is hoped the outcome of the present study will provide some direction for the more effective implementation of biofeedback therapy in the treatment of the TMJ syndrome and the alleviation of chronic pain.

II. REVIEW OF THE LITERATURE

A. TMJ/MPD

History of the TMJ/MPD Syndrome

In 1935, J. B. Costen, an otolaryngologist, published an article which presented the cases of 11 patients suffering from a variety of "ear and sinus" symptoms which he claimed were due to "disturbed function of the temporomandibular joint" (Costen, 1934). The symptoms he observed consisted of impaired hearing, a stuffy or stopped sensation in the ears, tinnitus, pain within and about the ears, dizziness, severe headache, and burning sensations in the throat, tongue, and nose. Costen attributed these symptoms to some disorder or irregularity in the anatomic function of the temporomandibular joints, their connective tissues and muscles due to a collapse in vertical dimension resulting from loss of posterior tooth support. In treating these patients, Costen claimed that these symptoms were relieved in nine of the eleven cases by prosthetic dentistry to open the bite.

Costen's work provided a theoretical link between these previously unrelated symptoms which had long puzzled researchers, and the direct mechanical function of the temporomandibular apparatus. In the decade and a half that followed Costen's article, however, a number of authors criticized his theory on the basis of clinical treatment outcome and anatomical research (Guralnick, Kaban, & Merrill, 1978). During this period Costen's original list of symptoms was modified as some were dropped as being unrelated to TMJ function, while others were added.

In the 1950's Laszlo Schwartz and his colleagues redefined "Costen's syndrome", as it was commonly known, and termed it the "pain-dysfunction syndrome" (Schwartz, 1955, 1956, 1958, 1959). Schwartz considered the symptoms relating to the TM-joint analogous to those surrounding other joints in the body. Pain in the temporomandibular area, as well as the other related symptoms, was attributed to incoordination of the muscles of mastication. Muscular dysfunction was followed by muscle spasm which in turn led to a "persistent pain-spasm cycle" (Guralnick *et al*., 1978). Schwartz also considered the patient's occlusion, dental history, and psychological predisposition to be significant contributing factors in the onset and development of the pain-dysfunction syndrome. At this point, a theoretical understanding of TMJ disorders which encompassed both anatomy and physiology as well as psychology, began to replace "Costen's Syndrome" with its purely mechanical perspective on facial pain.

A further development in the theory of temporomandibular joint pain came with the work of Laskin, who in the late 1960's proposed the myofascial pain-dysfunction (MPD) theory. This theory extended the Schwartz's concept by viewing masticatory muscle spasm initiated by muscular overextension, muscular overcontracture, or muscle fatigue, as the primary factor in the onset of pain (Laskin, 1969). The muscle spasm could be precipitated by a number of factors including malocclusion and muscle fatigue produced by chronic oral habits such as bruxing or clenching the teeth.

Symptomatology

From the complex of symptoms that constituted Costen's original syndrome, modern TMJ pain-dysfunction theory since the 1950's has retained only a few. Schwartz, in a well-known study of 491 TMJ patients (Schwartz, 1959), found that the three most prevalent symptoms were: pain (75%), clicking (9%), and limitation of mandibular movement (7%). Additionally, pain occurred in conjunction with other symptoms such as clicking and limitation in 62% of the sample. The quality of the pain was described as "a constant unilateral jaw ache, earache, or headache, usually aggravated by mandibular movement" (Schwartz & Chayes, 1966).

A few years later, Laskin clarified what were proposed to be the essential symptoms in the pain-dysfunction syndrome. As did Schwartz, Laskin emphasized the presence of unilateral pain, described as a constant, dull ache in or about the ear, which can extend generally to the head and neck. The pain is often worse in the morning although it can also augment during the course of the day (Laskin, 1969). Clicking and limitation of opening were also common complaints. To this symptom triad Laskin also added muscle tenderness, a condition of which most patients were not aware.

At present there appears to be general agreement upon the four principle symptoms relating to the pain-dysfunction syndrome: 1. Pain, predominantly unilateral, 2. Click, 3. Limitation of mandibular movement, 4. Muscle tenderness (Brooke, Stenn, & Mothersill, 1977). Other researchers have found additional symptoms to be associated with the syndrome. These include chronic minor illness such as migraine, back, neck or shoulder pain, skin disorders, hay fever, and asthma (Berry, 1969); recurrent headaches (Magnusson & Carlsson, 1978); stuffiness in the ear, hearing loss, dizziness and disequilibrium (Koskinen, Paavolainen, Raivio, M., & Roschier, J. 1980; Weinberg, 1980); tinnitus, blurred or double vision, and change of voice (Reade, 1984). However, for the most part, the symptoms most commonly accepted in the diagnosis of the TMJ syndrome continue to be those defined by Schwartz and Laskin.

Objective Clinical Manifestations

Temporomandibular joint pain-dysfunction disorders can be classified into two groups: 1. disorders primarily organic in nature and, 2. functional or non-organic disorders.

The first group comprises the category of TMJ dysfunction or disease proper, that is, disorders directly linked to organic changes or disturbances in the temporomandibular joint and its related structures. These can include displacements of the disk or condyle, inflammatory conditions and arthritis (Lamont-Havers, 1966; Bell, 1969), ankylosis, fractures, muscular dysfunction, and occlusal disharmony (McNeil, Danzig, Farrar, Gelb, Lerman, Moffett, Pertes, Solberg, & Weinberg, 1980). The onset of these conditions may be due to trauma as well as the adaptive changes, bone resorption and remodeling etc., that occur throughout the life cycle (Blackwood, 1966).

The second category pertains to the disorder commonly termed the "Myofascial Pain-Dysfunction" (MPD) syndrome, whose symptoms are primarily associated with masticatory muscle spasm and unilateral pain (Laskin, 1969). In the case of myofascial pain-dysfunction, there is no evidence of pain originating from organic changes or trauma to the TMJ itself. However, the distinction between the TMJ/MPD syndromes is still a point of debate in that, long-term muscular spasm can produce changes in occlusal harmony as well as degeneration of the temporomandibular joint. Alternately, organic or structural changes in the TMJ can also lead to muscular dysfunction. Rather than two separate and distinct entities, the present writer will view TMJ and myofascial pain-dysfunction as part of a continuum of polarized but often overlapping clinical signs and symptoms. Often, the distinction between the two syndromes will depend upon the severity, distribution, and duration of the symptoms as well as the timing of the examination.

In the literature there is presently a general consensus of opinion over the clinical signs that comprise the TMJ syndrome. These are: 1. pain and tenderness in the area of the TM joints and the muscles of mastication, 2. sounds (clicking, popping, crepitus) accompanying condylar movement and, 3. limitation of mandibular movement (Rugh & Solberg, 1979). The presence of joint sounds alone is not considered sufficient for determining TMJ dysfunction.

In addition to these primary signs, the clinician may also include a visual assessment of head and facial symmetery to screen for structural and/or soft tissue abnormalities such as muscular hypertrophy (Schwartz & Chayes, 1966); deviation of the mandible on opening and closing (Greene, Lerman, Sutcher, & Laskin, 1969); mandibular posture and occlusion (Weinberg, 1980); bruxism (Glaros & Rao, 1977); mouth breathing (Garry, 1982); referred pain (Travell, 1960; Campbell, *et al*., 1982), general postural abnormalities and limitation of movement in the neck, upper and lower back (Gelb, 1977; Gelb & Bernstein, 1983).

Developmental Sequence

The characteristics of the TMJ pain-dysfunction syndrome vary according to the age of the patient. Gelb (1977, 1980) presented the chronological sequence of the TMJ syndrome based on the Funt-Stack Index of the

Craniomandibular Pain Syndrome. Gelb's representation is as follows:

Ages 4-7 headaches; stuffiness and/or itching in the ears; earaches with no infection; grinding or clenching the teeth; *Ages 8-10* headaches; ringing sounds in the ears; back teeth sore; popping or clicking sound when mouth is opened or closed; *Ages 11-15* headaches; bloodshot eyes; curvature of the spine; roaring, buzzing and hissing sounds in the ears; dizziness; muscles surrounding jaw joints sore; back teeth sore; creaking sound when mouth is opened or closed; limited ability to open mouth; *Ages 16-20* headaches; pain behind the eyes; neck and shoulder pain; curvature of the spine; roaring and ringing sounds in the ears; dizziness; back teeth painful; jaw joints painful to move; *Ages 21-30* headaches; frequent sinuslike pain; facial features asymmetrical; pain behind the eyes; neck and shoulder pain; backaches; facial pains; muscle surrounding jaws sore and tired; *Ages 31-40* severe headaches; pain behind the eyes; chronic sore throat; shoulder pain mimicking bursitis; numbness in the arms; neck pains, backaches; facial pains; jaw joint becoming arthritic; *Ages 41-50* severe headaches; severe pain behind the eyes; numbness in the arms; incapacitating neck pain; incapacitating facial pain; osteoarthritic degeneration of the jaw joint; *Ages 51-60* compounding of almost all the previously mentioned symptoms; *Ages 61-70* loss of control over movement in face, tongue and lips; other associated symptoms (1980, pp. 44-45).

Epidemiology and Incidence Rate

The majority of epidemiological studies of mandibular dysfunction have been Scandinavian and have used varying sample sizes representing both patient and general populations (Reider, Martinoff, Wilcox, 1983). In a study of 1069 Swedish shipyard employees, Hansson and Nilner (1975) reported that 79% of the subjects manifested some TMJ of related muscular symptoms, and 30% had a combination of two or more clinical signs. TMJ clicking was the most frequently observed sign, occurring in 65% of the subjects. Following a review of epidemiological literature relating to mandibular dysfunction, Helkimo (1976) concluded that there did not appear to be any sex differences in the distribution of the disorder within the general population.

Conversely, in studies of clinical populations, many researchers report that between 65% and 80% of TMJ dysfunction patients are female, with the predomninant age group lying in the 20-40 year bracket (Moss & Garrett, 1984). In accounting for this divergence from the epidemiological data, some investigators maintain that women are generally more health conscious than men and tend to seek medical help for pain more readily (Carraro, Caffesse, & Albano, 1969; Agerberg, & Carlsson, 1972). Additionally, Reider proposes that women as a group are more sensitive to TMJ pain, sounds, and other symptoms than are men, in spite of the relatively equal distribution of these in the general population (Reider *et al*., 1983). Other authors have suggested that women are more prone to psychosomatic disease (Agerberg & Carlsson, 1972; Heiberg, Heloe, & Krogstad, 1978); and less tolerant of pain (Woodrow, Friedman, Siegelaub, & Collen, 1975). As a result of these findings, as well as the general preponderance of female TMJ patients, most studies of mandibular dysfunction have used primarily female subjects.

In summary, epidemiological studies of disorders of the masticatory system have concluded that signs and symptoms of mandibular dysfunction occur in a large percentage of the general population, although the incidence of pain or other discomfort warranting medical attention is relatively small. Also, there do not appear to be any sex differences in the distribution of these signs or symptoms. On the other hand, in studies of clinical populations, those who do seek assistance for problems associated with TMJ dysfunction, female patients are clearly in the majority.

Symptom Mechanisms and Etiological Factors

Pain is the most frequently encountered concern of patients presenting with TMJ pain-dysfunction (Butler, Folke, & Bandt, 1975; Nel, 1978). The pain is usually unilateral in cases of MPD, and commonly bilateral in instances of organic temporomandibular joint dysfunction (Weinberg, 1980). The quality of the pain has been described in varying terms, from a dull aching to a severe, excruciating pain (Weinberg, 1980). One author reports some of his patients relating a feeling of "worms in the bones" and of "teeth growing up into the head" (Alling, 1981).

The quality of pain experienced by the patient is significant in making a proper diagnosis of the particular type of dyfunction. Bell (1982) has categorized orofacial pains into four main groups: a. superficial somatic pain, b. deep somatic and visceral pain, c. neurogenous pain, and d. psychogenic pain. Both superficial and deep somatic pain are the result of noxious stimulation of body tissues containing normal innervation. Pain in this category can reflect involvement of muscles, joints, connective tissue, or the bones; and can be referred as well as localized. Burning or throbbing pain which is aggravated by palpation or movement may indicate inflammation or muscle spasm. Neurogenous pain is characterized by the presence of paroxysmal shooting pains extending along the course of a particular nerve. Psychogenic pain is often poorly defined and wandering although it can mimic other types of pain. Emotional disturbances often intensify the pain, or serve as the primary etiological factor as in conversion hysteria pain. The pain experienced by the TMJ pain-dyfunction patient is most often of the deep somatic type involving the masticatory musculature and/or joint structures.

Etiological theories accounting for the pain mechanisms in mandibular dysfunction can be roughly classified into two groups: a. the mechanical displacement theory, b. the muscle dysfunction theories.

Mechanical Displacement Theory

Proponents of the mechanical displacement theory maintain that pain results from condylar displacement due to mandibular overclosure. Loss of posterior occlusion is the primary precipitating factor. Costen's original theory viewed the observed complex of "ear and sinus" symptoms as resulting from pressure applied by the condyle on the auriculotemporal and chorda tympani nerves as well as that transmitted through the temporal bones of the skull (Costen, 1934; Moss & Garrett, 1983). Further anatomical research, however, discounted Costen's nerve compression explanation for facial and head pain (Zimmerman, 1951). Recently, other investigators have proposed that pain may result from posterior and superior condylar displacement impinging upon, and causing inflammation of, the highly vascular and innervated connective tissue of the retrodiscal pad, the posterior portion of the meniscus which attaches to the posterior wall of the joint capsule (Weinberg, 1979a, 1979b; Hylander, 1980; Moss & Garrett, 1984).

From this theoretical point of view, increased activity in the masticatory muscles is not the primary etiological factor in the pain-dysfunction syndrome, rather, the responsibility rests with faulty or disturbed condylar/occlusal relationships. In such cases, because of improper condylar position or faulty occlusion, the forces of the musculature are not adequately buffered and act directly upon the temporomandibular joint leading to pain and dysfunction (De Boever, 1973). The condition may also be aggravated by the presence of chronic oral habits such as bruxism or continued clenching, gum chewing, pipe smoking, and mouth posture required for playing various musical instruments.

Clicking and other joint sounds accompanying mandibular movement are thought to be due to posterior displacement of the condyle in relation to the articular disk. Crepitus and other grating or scraping sounds are usually associated with perforation of the mensicus and are a sign of advanced joint dysfunction and possible degenerative changes (Weinberg, 1980).

Limitations in mouth opening and deviations during mandibular movement are also often interpreted as a result of faulty condyle/disk function, with the disk being displaced anteriorly and wedged between the condyle and the articular eminence thus preventing the normal translatory motion of the joint (Moss & Garrett, 1984). This type of displacement of the disk can occur bilaterally or unilaterally. In the former instance, restriction of movement results; in the latter, mandibular deviation will be apparent. Other explanations for limitation reviewed by Moss & Garrett include organic changes such as ankylosis due to fibrous adhesions or calcifications of the articular surfaces, inflammation of the capsular ligaments, and masticatory muscle spasm.

Muscle Dysfunction Theories

Muscle dysfunction theories of the TMJ pain-dysfunction syndrome maintain that pain results from masticatory muscle hyperactivity and spasm. These fall into two major groups: 1. Neurophysiologic theories and, 2. Psychophysiologic theories.

1. Neurophysiologic theories of the TMJ syndrome postulate abnormal occlusal relationships which lead to altered proprioceptive impulses and inappropriate muscle activity. Such activity can take the form of increased muscle tonus, spasm, clenching and bruxism (Ramfjord, 1961a, 1961b), and is linked to a background of psychic tension, anxiety, and stress (De Boever, 1973). The excessive muscle activity may lead to pain in the muscles, ligaments, and the temporomandibular joint through abnormal mandibular posture.

Other signs and symptoms of dysfunction, such as mandibular deviation and limitation, are due to muscle splinting and spasm which inhibit mandibular movement on the affected side. Deviation will then occur toward the dysfunctional side.

2. Psychophysiologic theories of TMJ pain-dysfunction are based on the concept that pain is primarily the result of increased masticatory muscle activity, incoordination, and spasm intimately related

to stress response and other psychological factors.

Schwartz and his colleagues were the first to advance the theory that psychological factors such as stress, nervous tension, anxiety, etc. play a key role in the onset, maintenance, and exacerbation of temporomandibular joint pain-dysfunction (Schwartz, 1955, 1956; Schwartz & Cobin, 1957). From this point of view, the patient's response to stress takes precedence over even severe malocclusion in the development of the syndrome (Greene, 1980). On the other hand, the psychophysiological theory refers to acute malocclusion produced by changes in muscle balance. This symptom develops quickly and usually subsides when the muscular problems are resolved. Occlusal restorations and other equilibration procedures are contraindicated, although they may be necessary if the muscle imbalance is not quickly rectified and the occlusion shifts to restore a proper functional relationship.

The psychophysiological theory was further developed by Laskin who, in 1969, advanced the theory of the myofascial pain-dysfunction syndrome. As with Schwartz, Laskin maintained that masticatory muscle spasm is the primary cause of pain and the other signs and symptoms of the the pain-dysfunction syndrome. Spasm can be initiated in one of three ways: 1. muscular overextension, 2. muscular overcontraction, or 3. muscle fatigue (Laskin, 1969; Mikhail & Rosen, 1980). Laskin

(1980) also includes trauma as a possible etiological factor in muscle spasm.

Although muscle spasm may be intiated by mechanical dental factors such as improper dental restorations, prostheses, malocclusion, and alveolar bone resorption, Laskin believes that most cases of the pain-dysfunction syndrome are due to muscular fatigue produced by muscular hyperactivity. This hyperactivity can take the form of "chronic parafunctional habits" including clenching and bruxing the teeth, habits he considers to be an "involuntary tension-relieving mechanism in response to psychological stress" (Laskin, 1980). Clark, Beemsterboer, and Rugh (1981) documented the direct link between pain-dysfunction symptoms and nocturnal muscle activity (bruxism) by monitoring masseter EMG levels in 85 subjects with varying degrees of jaw dysfunction. A significant correlation was found between the amount of nocturnal masseter activity and the clinical manifestations of mandibular dysfunction.

Temporomandibular joint sounds such as clicking and popping are caused by muscle incoordination rather than condylar displacement, while crepitus and other grating sounds reflect actual joint pathology (Laskin, 1980). Limitation and deviations in mandibular movement are primarily due to muscle spasm as is the muscle tenderness to palpation characterisic of pain-dysfunction patients.

Psychological Implications

With the Psychophysiologic pain-dysfunction theory maintaining that stress, anxiety, and other emotional and personality factors play a significant role in the onset and maintenance of the syndrome, it is not surprising that the psychology of the TMJ patient has been the subject of many studies in the last thirty years. Rugh and Solberg (1976,) have reviewed and classified them according to their predominant theoretical orientation. The most significant of these to consider here are: (a) psychoanalytic concepts, (b) personality traits, and (c) learning theory.

Psychoanalytic Concepts

One of the first psychological investigations in this area was conducted in the mid 1950s by Moulton, a psychoanalytically oriented psychiatrist and associate of Schwartz. After a psychiatric study of 35 TMJ patients, Moulton concluded that all but four manifested signs of psychological disturbance. Twenty were evidently anxious and nervous, 18 of the 35 either bruxed or clenched their teeth, presumably when repressing emotion, especially anger. Furthermore, 11 of the patients were diagnosed as psychotic or prepsychotic (1955b). In another study, Moulton linked the pain-dysfunction syndrome to hysterical conversion reaction of repressed sexual guilt (Moulton, 1955a). The conclusions of Moulton's work suggest that TMJ pain-dysfunction patients tend to exhibit various personality and emotional disorders characterized by increased muscular response to emotional and psychic stress.

Lefer (1966) concluded that the TMJ patient usually has "poor ego boundaries and utilizes bodily reactions to diminish the level of anxiety aroused by the threatened eruption into awareness of oral sadistic, extractive, and incorporative needs" (p. 149). Furthermore, these patients often see their dentists as authority figures with whom they form symbiotic relationships to protect themselves against feelings of bereavement from threatened or actual loss of the mother (Lefer, 1971).

Fine (1971), in a larger study involving 50 TMJ patients and 50 non-TMJ controls, concluded that 76% of the experimental group subjects manifested psychiatric symptoms. The most frequently observed disorders were characterized as depressive-anxiety reactions initiated by bereavement experiences and other emotional stressors. Fine thus concluded that psychological factors play a more important role in the onset and development of the mandibular pain-dysfunction syndrome.

In sum, psychoanalytic concepts hold that symptoms of TMJ dysfunction arise primarily out of denial or repression of anxiety, conflicts, or emotional needs and impulses, as well as past or present experiences involving bereavement, frustration, or guilt.

Personality Traits

Most personality studies of TMJ pain-dysfunction patients have used structured interviews and/or written personality inventories. The most frequently used assessment instrument has been the Minnesota Multiphasic Personality Inventory (MMPI) which has generally revealed that pain-dysfunction patients tend to have personality profiles similar to those of patients suffering from a wide variety of other psychophysiologic disorders (Olson, 1980). Also, like these other patients, TMJ pain-dysfunction patients often suffer from related psychophysiologic disorders as reported by Berry (1969) and Lupton (1966).

In one of the earliest studies of the "TMJ personality" using the MMPI, supplemented by the Cornell Medical Index and the Edwards Personality Profile, Kydd (1959) found that 23 of 30 TMJ subjects (76%) evidenced signs of significant emotional disturbance and presented as anxious, tense, apprehensive, and overreacting to pain. McCall, Szmyd, & Ritter (1961) administered the MMPI to 70 TMJ patients and two non-TMJ control groups of 70 subjects each. Following an analysis of the inventory results, it was possible to discriminate between the TMJ and the control groups on the basis of 48 significantly different responses. Of these TMJ specific items, 22 included somatic complaints, while the remainder were associated with chronic anxiety, worry, and other miscellaneous factors. McCall *et al.* were not, however, able to define a particular personality profile from their data, although they anticipated this would eventually be possible with the development of better research instruments. Other personality studies have produced equally inconclusive results, some investigators report definite TMJ personality characteristics, while others fail to find any group personality tendencies.

In a study of 37 female TMJ patients using the MMPI, Interpersonal Adjective Checklist, and the Thematic Apperception Test (TAT), Lupton (1966, 1969) described the group as rigid and hypernormal. These women were perceived as being matriarchal, dominant, responsible, generous, managerial, and narcissistic; they also seemed to rely heavily upon denial and repression in order to maintain a consistent self-concept.

Similar personality trends were reported by Molin, Schalling, and Edman (1973) who found that, compared with a normal group, the TMJ sample rated higher scores in neuroticism, somatic anxiety, muscular tension, aggression and superego strength. They also had a tendency to be more conscientious, responsible, orderly and serious. Schwartz, Greene, & Laskin (1974) also found that pain-dysfunction patients obtained elevated scores in the "neurotic triad" of the MMPI (the hypochondriasis, depression and hysteria scales). In a multiprofessional Scandinavian study of 113 MPD patients and 46 control subjects, Heloe, Heiberg and Krogstad (1980) concluded that pain-dysfunction patients tend to "over control" their emotions, particularly aggression. However, both groups scored high on the MMPI Anxiety scale.

On the other hand, Solberg, Flint, and Brantner (1972), also using the MMPI on 29 TMJ patients with an equal sized control group, reported no common personality traits or patterns. Some of the patients demonstrated greater anxiety, but this was viewed as within the range of a normal personality profile.

On the question of a "TMJ personality", Rugh & Solberg (1979) conclude that there does not appear to be any one personality trait or characteristic common to all pain-dysfunction patients. Rather, TMJ patients may display a variety of personality characteristics, which may or may not render them suceptible to the syndrome. Unless future research can utilize more sensitive assessment procedures and define more adequate personality categories, Rugh & Solberg consider inquiry in this area to be a futile pursuit.

Learning Theory

From a learning theory perspective, pain behavior, like any other behavior is learned primarily in a social context as a result of observation and modeling (Craig, 1983). The child observes various modeled behaviors and the consequences that ensue. Those behaviors that are followed by some form of positive reinforcement will tend to be assumed by the child as socially correct or acceptable. In this way, attitudes toward pain and actual pain behavior itself are transmitted from parent to child (Melzack, 1973). Following this model, TMJ pain-dysfunction can also be viewed as a learned behavior occurring in reponse to certain stimuli and contingent upon subsequent positive reinforcement.

Fordyce (1976) has distinguished between two categories of pain: (a) respondent pain, and (b) operant pain. Although there has not been an extensive analysis of the TMJ pain-dysfunction syndrome from this perspective, the concepts of respondent and operant pain offer important insights into the psychological dimension of the syndrome.

Respondent pain is controlled by specific stimuli and invariably follows presentation of the stimuli. Operant pain, although it may also be a response to antecedent stimuli, is primarily controlled by its consequences. That is, operant pain behavior is maintained by positive or reinforcing consequences; conversely, operant pain behavior will diminish if not followed by some type of reinforcement (Fordyce, 1976).

It is evidently not realistic to attempt to place TMJ or facial pain into one or the other category, rather the syndrome may include elements of both respondent and operant pain. The present discussion will deal only with the concept of operant pain and its implications for understanding the TMJ pain-dysfunction syndrome.

The concept of operant pain as it relates to TMJ pain can be viewed in three categories as set out by Fordyce (1976):

1. Direct and positive reinforcement of pain behavior, where the behavior persists because of some continued intrinsic reward or benefit that results. The TMJ pain-dysfunction literature is replete with accounts of patients whose symptoms and complaints dominated their family and personal lives, exhausting family finances and local health services. A careful behavioral analysis of these patients, their families, and the relationship they have with health professionals suggests that there is a great resemblance between TMJ pain-dysfunction sufferers and patients with other common pain syndromes (Rugh & Solberg, 1976; Marbach & Lipton, 1978). The communicative and manipulative aspect of such pain is one of its key reinforcers and operates in the form of the increased personal attention, sympathy, concern, etc. the patient receives from his significant others (Sternbach, 1974). Other examples of intrinsic reinforcement include medication schedules which use pain as the criterion i.e., "take only as

needed" and rest breaks determined by the presence of pain (Fordyce, 1983).

2. Indirect but positive reinforcement of pain behavior by avoidance of aversive consequences, whereby pain excuses the patient from difficult personal, emotional, or social situations. From this perspective, TMJ pain may have, at some point in the patient's history, provided relief or time out from some aversive circumstance. The aversive situation that was avoided in the past does not have to repeat itself to again elicit the same type of pain behavior, any difficult, disagreeable, or otherwise unpleasant situation serves equally well. Thus the pain-dysfunction behavior persists (Fordyce, 1976).

3. Failure of well behavior to receive positive reinforcement; in other words, operant pain behavior is much more likely to develop and continue than is operant well behavior. Fordyce cites the example of an individual of limited talents and skills for whom displays of well behavior imply equivalent displays of well performance and competence. As he is not likely to be very successful in the range of what would be considered normal daily functioning, this individual receives very little, if any, reinforcement for his efforts. "Such a person," Fordyce continues,

> can be expected to develop an extensive repertoire of behaviors designed both to avoid

> the aversive consequences of failure and to elicit positive reinforcement from the immediate environment. . . . If practiced long enough, such behavior may lead others to lessen their demands and expectations . . . . The person might have adopted a reclusive, hostile, isolated style designed to keep people (and their performance demands) at a distance. But he might also have developed a vast illness repertoire. Weakness, ease of fatigue, hypersensitivity to pain and stress, and a readiness to get sick in the face of demands may describe much of the behavior repertoire (1974, p. 69).

The learning theory concepts of operant pain behavior described above are only offered as possible psychological mechanisms functioning in the personalities of some TMJ patients. The notion of operant pain behavior cannot be over generalized to explain all of the TMJ pain-dysfunction syndrome any more than other pain syndromes, but it does provide an interesting and useful perspective on the psychogenic nature of TMJ and other facial pain.

Treatment

Various types of treatment strategies have been applied to the patient diagnosed to have TMJ pain-dysfunction syndrome, from psychotherapy to surgery--each theoretical perspective on the etiology of the syndrome necessarily promotes a particular treatment orientation. The present review will consider the principle treatment strategies that have evolved out of the mechanical displacement and muscle dysfunction theories. As with some of the etiological conclusions reached by these two theoretical outlooks, the resulting treatment strategies may overlap, but they do so for different reasons. For the sake of simplicity, the different types of treatment procedures can be placed in two categories: (a) dental approaches, and (b) psychological approaches.

Dental Approaches

Three basic dental treatment approaches are commonly used in the management of TMJ pain-dysfunction syndrome: occlusal splints, equilibration procedures, and prescribed medications (Moss & Garrett, 1984).

Occlusal splints perform various tasks among them: restoring proper condyle/fossa relationships by modifying the position of the mandible, redistributing the non-functional forces of bruxing or clenching more evenly over the entire occlusion and its musculature, and relieving muscle tension or hyperactivity by altering proprioceptive signaling (Carraro & Caffesse, 1978; Kawazoe, Kotani, & Yamada, 1980); and increasing the resting length of the elevator muscles, thus reducing their isometric tension (Christensen, 1980).

B

Occlusal splint therapy is often followed by equilibration procedures such as selective grinding, restorations, and orthodontics. The aim of these procedures is to permanently reproduce in the occlusion the stabilizing effects of the previous splint by restoring optimal occlusion and mandibular relationship.

The use of splint therapy and occlusal equilibration has been widely used, with reasonable success (Mejersjo & Carlsson, 1983). However, some of the inherent disadvantages are the long-term nature of treatment in the case of occlusal splints, and the cost and irreversibility of occlusal equilibration.

In a recent article reviewing long-term results of TMJ treatment in 151 patients, Mejersjo and Carlsson (1983) reported an 80% success rate using primarily occlusal adjustment, splint therapy, and therapeutic exercises. Greene and Laskin (1983) also reported success rates in the 80% range for various treatment modalities. This contrasts with Beard and Clayton (1980) who found 100% reoccurence of symptoms in patients after discontinuing splint therapy.

Other studies have also considered the role played by the placebo effect in successful treatment. Greene and Laskin (1972) found that, after a treatment period of 2-6 weeks, 28 of 71 patients had improved with only the use of a non-occluding placebo splint. In another study Goodman, Greene, and Laskin (1976) reported that

16 of 25 patients (64%) experienced complete or nearly complete remission of their symptoms after having received mock equilibration consisting of selective grinding and "adjustment" of non-occluding tooth surfaces. Clarke (1982) also maintained that the success of occlusal therapies is primarily due to the placebo effect.

Commonly prescribed medications used in the treatment of the TMJ pain-dysfunction syndrome include analgesics and anti-inflammatory agents, muscle relaxants, steroids, antivertiginous drugs, and vitamins and minerals (Hall, 1982). In another placebo study, Greene and Laskin (1971) compared the effect of meprobamate, a tranquilizer and muscle relaxant, with a placebo drug. The authors found that 58% of the patients administered meprobamate reported improvement, as did 31% of the placebo patients. Laskin and Greene (1972) also reported an extension of their research in a study which evaluated the influence of the doctor-patient relationship on placebo drug therapy for MPD patients. In this project, 50 MPD patients were prescribed a placebo drug accompanied by precise dosage intructions and assurances of its effectiveness. At the end of the four week experimental period, twenty-six of the fifty patients (52%) reported some improvement in their condition with eight not requiring further treatment.

Psychological Approaches

A number of psychological approaches have been used in treating TMJ patients. From the mid 1950s with the discovery of psychological factors operating in the syndrome, psychotherapy in the form of individual counselling or group therapy has been prescribed (Moulton, 1955b; Kydd, 1959; Lefer, 1966; Lupton, 1969; Pomp, 1974; Marbach & Dworkin, 1975; Kopp, 1979). Hypnosis has also been utilized for pain and symptom control (Tarte & Spiegel, 1977), as has acupuncture (Corcos & Brandwein, 1976; Quint, 1982).

Another frequently used therapy procedure stemming from the muscle hyperactivity theory is that of relaxation training, based primarily on Jacobson's Progressive Relaxation technique (Gessel & Alderman, 1971; Reading & Raw, 1976; Raft, Toomey, & Greg, 1979). In the early 1970s biofeedback training began to be used as an adjunct to traditional progressive relaxation therapy. Since that time biofeedback has emerged as one of the foremost psychological therapies in the treatment of TMJ pain-dysfunction disorders. In the following pages the theoretical basis for biofeedback therapy and the extent of its application in the syndrome will be reviewed.

B. BIOFEEDBACK AND THE TMJ PAIN-DYSFUNCTION SYNDROME

Rationale for Biofeedback Therapy in TMJ

The Concept of Biofeedback

Fundamental to the concept of biofeedback is the discovery that voluntary and involuntary or autonomic physiological processes can be influenced or controlled by volition. Basmajian (1979) described biofeedback as:

> the technique of using equipment (usually electronic) to reveal to human beings some of their internal physiological events, normal and abnormal, in the form of visual and auditory signals in order to teach them to manipulate these otherwise involuntary or unfelt events by manipulating the displayed signals. This technique inserts a person's volition into the gap of an open feedback loop--hence the artificial name biofeedback . . . (p. 1).

Implicit in the theory of biofeedback is the assertion that any physiological process which can be monitored and meaningfully "fed back" to the individual can be willfullly controlled or altered by the individual himself. In other words, the subject has the capacity to self-regulate much more than was traditionally believed. Additionally, some authors maintain that every change in a physiological state is accompanied by a corresponding change in emotional or psychic state and vice versa (Green, Green, & Walters, 1970). Consequently, biofeedback has been used to treat disorders which are both physiologic and psychosomatic in nature.

By its emphasis on self-regulation, biofeedback breaks the traditional doctor-patient role: the ultimate responsibility for change rests with the patient and his level of motivation. Pelletier (1977) writes that:

> Biofeedback is one of many approaches in the revolution of psychotherapy and medicine which places the responsibility of illness, health, and especially personal growth upon the individual. In this evolving model, the therapist acts as a guide or a teacher in order to maximize the conditions for the patient's growth and self-healing (p. 266).

The Application of Biofeedback

One of the most frequent applications of biofeedback is in the treatment of psychophysiologic or psychosomatic disorders. Such disorders are characterized by the complex interplay of psychological factors and physiological responses in the onset or maintenance of the disorder and its clinical signs and symptoms. Tarnopolsky and McLean (1976) describe psychosomatic illness as occurring when "physical predisposition, psychological threat and a psychological dynamic vulnerability complementary to that threat are found in the same individual at the same time" (p. 96). Biofeedback, then, is a means of regulating or modifying the interaction of the above physical and psychological factors with a view to limiting and, ultimately, arresting the illness cycle.

The place of biofeedback in the context of TMJ therapy is best seen in relation to the muscle hyperactivity etiological theory of the pain-dysfunction syndrome. As Schwartz and later, Laskin, have maintained, the pain syndrome arises primarily out of masticatory muscle dysfunction, primarily consisting of abnormally elevated muscle tension and/or spasm. Jacobson (1967) has written that suffering and pain is physiologically related to increased muscle tension; as the patient relaxes to a near zero level of activity, pain responses diminish or disappear. Jacobson notes, however, that pain does not appear to diminish proportionally with muscle tension, but only when total relaxation is achieved. One of the goals, then, of biofeedback training in the TMJ pain-dysfunction syndrome is to enable patients to reduce their masticatory muscle tension, or hyperactivity as much as possible while maintaining general overall relaxation.

While the muscle hyperactivity characteristic of the TMJ pain-dysfunction syndrome can result from mechanical or physical irregularities such as trauma or occlusal imbalances, this theory maintains that muscle hyperactivity is more often due to psychophysiological causes, primarily related to the patient's response to stress, environmental as well as inner psychic stress. These two types of stress can be viewed as (a) physical stress and, (b) psychological stress.

Haber, Moss, Kuczmierczyk, and Garrett (1983) define physical stress as a state of discomfort resulting directly from some physical activty or event. Psychological stress, on the other hand refers to the discomfort arising out of the subjective interpretation of some event. The authors add, however, that the two types of stress are often related and both may be implicated in the pain-dysfunction syndrome.

In a more comprehensive understanding of the link between stress and the TMJ syndrome, Selye's (1956) formulation of the *general adaptation syndrome* (G.A.S.) offers valuable insight. Selye defines stress as "the state which manifests itself by the G.A.S." that is, "adrenal stimulation, discharge of lymphatic organs, gastrointestinal ulcers, loss of body-weight, alterations in the chemical composition of the body. . ." (p. 47). The G.A.S. is accompanied by a *local adaptation syndrome* (L.A.S.) in the tissues more directly affected by stress. This response consists of the production of adaptive hormones, namely, anti-inflammatory hormones which inhibit excessive defense reactions; and proinflammatory hormones which stimulate defense reactions. The activity of these hormones within the region of the L.A.S., in this case the temporomandibular apparatus, may lead to the emergence of the TMJ syndrome such as muscle dysfunction and spasm, arthritic changes in the joint and general inflammation of related structures. From this perspective the TMJ syndrome could be understood as what Selye terms a "disease of adaptation" (p. 66) where muscular hyperactivity or adaptive changes arise out of the inability of the TMJ system to adapt adequately to the demands placed upon it by the exigencies of daily life.

As Haber *et al.* (1983) indicate, stressors can be physical and/or psychological in nature. Consequently, the role of biofeedback in therapy for patients with TMJ syndrome is two-fold and can be summarized as follows: 1. Biofeedback serves to monitor and reduce overall masticatory muscle tension and correct muscular imbalances due to chronic oral habits, posture, etc. (physical stress). 2. Feedback can make the patient aware of the role his own affective and cognitive responses to life events (psychological stress) play in maintaining dysfunctional muscle activity.

Review of Biofeedback Application in the TMJ Syndrome

The use of biofeedback in dentistry is a relatively recent development. Over the last ten to fifteen years it has gone from occupying a tentative research role to actual routine application in the treatment of various stress related dental disorders. Two excellent multidisciplinary works (Gelb, 1977; Morgan, House, Hall, & Vamvas, 1982) have devoted entire chapters to the application of biofeedback in the treatment of TMJ disorders. While not yet extensive, an interesting body of research and clinical literature has accumulated reporting the use of biofeedback in temporomandibular pain-dysfunction. Some of the key developments will be reviewed here.

A large number of studies have been published which discuss the link between psychological stress and increased masticatory muscle activity. Copeland (1954), presented 80 case studies of mandibular joint dysfunction in which abnormal muscle tension related to temporary "emotional disturbances or mental anxieties" was a significant etiological factor. On the basis of self-reports and clinical examinations of 900 patients, Franks (1965) determined that emotional causes were clearly implicated in bruxism and mandibular dysfunction. He did not find any significant differences between men and women in the incidence of bruxism.

In another early investigation into the effects of psychological stress on masticatory muscle tension, Perry, Lammie, Main, and Teuscher (1960) submitted eight dental students to an identical stress situation and observed their pre and post stress levels of masseter and temporalis EMG activity. From this study the authors concluded that:

1. Electromyography is useful in determining states of anxiety, and tensions in the muscles of mastication.

2. Immediate stress situations produce increased muscular activity in individuals with probable predisposing long-term emotional stress build-up.

3. Clinical and anatomical evidence exists for the often described tensions and aches within the masticatory musculature of individuals under emotional stress.

In a continuation of the work of Perry *et al.* (1960), Yemm (1968) subjected thirty subjects to two kinds of stressors; one where the subjects were to squeeze a pressure bulb to maintain a predetermined pressure, and the second, where subjects were to respond to a column of randomly flashing lights by pressing the corresponding buttons arranged in a horizontal row. In both tasks, speed and accuracy of response were monitored with instantaneous feedback given to the subjects in the case of incorrect or hesitant responses. Yemm found that, in the bulb squeezing test, masseter muscle activity was high before the task, increased yet further during, and subsided to the lowest level after the task. In the flashing light test, peak levels of muscle activity coincided with incorrect responses and presentation of the error signal. Yemm also observed a proportional relationship between the number of mistakes made and the magnitude of the muscle activity. Yemm concluded that masseter and temporalis muscle activity increases under stress.

Similar studies conducted by Yemm (1969a, 1969b, 1969c, 1971) reinforced this conclusion and added the significant observation that while TMJ dysfunction patients responded to stress by increasing masseter muscle activity, unlike normal subjects, their response over the course of the experiment did not diminish. Yemm postulated that pain-dysfunction patients are poor at adapting to stress, whether experimental or everyday; this could lead to continued and prolonged loading of the masticatory system, resulting in eventual dysfunction (1969b).

In a study of the effects of anxiety and frustration on masticatory muscle tension, Thomas, Tiber, and Schireson (1973) induced experimental anxiety and frustration in a group of TMJ patients and in equivalent non-TMJ controls. The authors found that only the TMJ group responded to the experimental anxiety with increased masseter and temporalis muscle activity, however, both groups responded to the frustration condition with the TMJ group demonstrating a significantly higher response than the control group. It appeared that, as a psychological stressor, frustration plays a more important role in the TMJ syndrome than does anxiety.

Support for the physical stress concept comes from the studies of Christensen (1971, 1975, 1979, 1981a, 1981b). Christensen reported that experimental maximum clenching and grinding in normal subjects produced pain similar in location and intensity to that experienced by MPD patients. The other signs of dysfunction, (tenderness to palpation, joint sounds, limitation of movement) were not found.

The work of Perry *et al.*, Yemm, and Christensen provide a basis for the application of biofeedback in the treatment of the TMJ pain-dysfunction syndrome. With the demonstration that pain-dysfunction patients typically exhibit masticatory muscle hyperactivity, and that part of the overactivity may be related to stress response, the theoretical groundwork was laid for the emergence of comprehensive biofeedback therapy.

Solberg and Rugh (1972) published the first paper describing a clinical application of biofeedback in dentistry. In this study, fifteen TMJ patients were given small EMG units to wear during the day. These units continuously monitored masseter muscle activity and whenever it exceeded a preset threshold level, a warning tone was emitted by the unit. The patients found that they typically clenched or bruxed their teeth when confronted with stressful situations. As a result of the self-monitoring the patients were able to identify such situations, avoid them, or find other ways of coping with the stress. Upon completion of the study, ten of the fifteen patients reported a significant improvement in their condition.

Mulhall and Todd (1975) reported the case of a 32 year old male patient who also was supplied with a portable EMG unit. The unit was set with a variable sensitivity threshold which was gradually adjusted in order to progressively shape the patient's awareness and frequency of clenching. After six weeks of self-monitoring, the patient returned the unit claiming an improvement in symptoms.

In another important research project, Budzinski and Stoyva (1973) compared the effects of visual feedback, auditory feedback, irrelevant feedback, and no feedback in reducing masseter muscle tension in eighty subjects. Although all participants were given written instructions to relax all muscles as much as possible, significant reduction in masster EMG was observed only in the meaningful feedback groups. The authors concluded that biofeedback training is a potentially useful therapy in the treatment of bruxism and TMJ dysfunction.

Carlsson, Gale, and Ohman (1975) reported one of the first applications of biofeedback in the treatment of TMJ dysfunction. The patient was a twenty-one year old woman described as having long-term pain in the TMJ, right lower jaw, and the masseter. After 18 sessions of weekly, or bi-weekly masseter biofeedback training, the patient was able to control her masseter muscle tension, and her pain. At time of writing, the authors reported that the patient had been totally painfree for six months.

In a similar case report, Carlsson and Gale (1976) reviewed the progress of a 59 year old woman whose primary complaint was severe pain in the left masseter. During baseline recordings obtained during relaxation, the left masseter showed more than five times the activity of the right as well as spontaneous, unilateral contractions. After nine weekly biofeedback sessions the patient reported a significant relief in pain, with no relapse after a one-year period.

Gessel (1975) conducted the first large-scale study using biofeedback as a treatment for facial pain. In this study, 23 MPD patients were given temporalis and masseter feedback for at least six thirty minute sessions and required to practice at home for one half hour daily. Those that did not improve after six sessions were considered failed and transferred to the second phase of the experiment, in which an anti-depressant drug program was initiated. Of the 23 patients, 15 reported satisfactory improvement of their symptoms after an average of five biofeedback sessions. On the basis of the pretreatment interview in which each of the participants had been rated in terms of high social drive, covert and overt depression, and social disability, Gessel found that the depression indicators discriminated between the patients responsive to biofeedback and those who derived no benefit from the training. Those who failed to respond to either treatment manifested signs of severe depression.

In another study using 24 MPD patients (Dohrmann & Laskin, 1976; 1978), 16 experimental subjects received nine sessions of auditory feedback from the masseter muscle. The remaining eight patients were assigned to a control group and told they would be receiving muscle relaxing electrical stimulation from the electrodes. At the end of the ninth session only two patients in the experimental group still complained of pain, as compared to four in the control group. Additionally, the experimental group showed a mean increase of 8.5mm in maximum opening without discomfort, compared to 2mm in the control group. The percentage of patients experiencing muscle tenderness upon palpation also decreased significantly in the experimental group (from 81% to 43%), but not in the control group (80% to 75%). At a twelve month follow-up, 12 of the 16 experimental patients claimed to be symptom free. The authors concluded that biofeedback is effective in treating the MPD syndrome.

Berry and Wilmot (1977) reported the results of a clinical trial of biofeedback with 35 mandibular pain-dysfunction patients. Each of the patients received an average of three biofeedback sessions. Although the authors did not state the length or frequency of the training sessions, 61% of the patients were relieved of symptoms within six months, and 85% within a year. One of the major benefits of the biofeedback, in the authors' view, was the increased awareness of masticatory muscle tension reported by all of the participating patients.

Another clinical application reported by Clarke and Kardachi (1977) used nocturnal biofeedback supplied by a portable EMG unit to modify seven patients' bruxism. The feedback signal did not awaken the subjects, but apparently served to rouse them to a lighter stage of sleep. During the treatment period, patients used the biofeedback unit nightly for one to four weeks after which reduction of symptoms was reported. The only exception was the seventh patient who used the EMG unit irregularly because of the inconvenience involved, she did not derive relief from either biofeedback or other forms of therapy. The authors concluded that biofeedback used in conjunction with some form of counselling or other psychotherapy is an appropriate form of treatment for the pain-dysfunction syndrome.

Olson (1977) described a study in which 15 MPD patients who had proven non-responsive to drug and splint therapy were randomly divided into three groups. Group 1 (5) received masseter EMG feedback from the painful side, group 2 (4) received frontalis feedback, and group 3 (6) was given both frontalis feedback and psychotherapy. The participants attended twelve biofeedback sessions and most attained a low level of muscle activity within the first six. On comparing pre and post-treatment symptom evaluations, Olson found that group 1 had benefitted from a slight decrease in pain but not in muscle tenderness, joint sounds or limitation, one patient in group 2 experienced complete remission, but there was no change in the others, two patients in group 3 reported complete remission of symptoms, 3 had little or no pain, and one was unchanged. Olson concluded that the combination of biofeedback and psychotherapy is more effective than biofeedback alone; biofeedback is not as effective with non-responding patients; feedback from the masseter may be more effective than from the frontalis.

Peck and Kraft (1977) reported a clinical trial of biofeedback training with 18 tension headache, eight back and shoulder pain, and six female TMJ pain patients. All the subjects were given two half-hour biofeedback session per week. Although EMG levels dropped across all groups, there was a reduction in pain only in the headache group; the back and TMJ groups did not appear to benefit from the biofeedback. The authors did not indicate the type and severity of clinical signs in the TMJ patients, nor did they report the amount of medication being taken, although this was monitored throughout. Finally, there was no mention made of any type of concurrent home relaxation practice during the period of training. Although the subject sample was small, the authors concluded that biofeedback training is not as effective in the treatment of TMJ and back pain as for tension headache.

In a more recent study, Manns, Miralles, and Adrian (1981) combined audiostimulation--pure tones of constant frequency and duration selected by the patient--and EMG biofeedback in the treatment of thirty-three bruxism and MPD patients. The subjecs were classified into two groups: Group I (14) whose symptoms had been present for a year or less and, Group II (19) whose symptoms were of more than a year's duration. Audiostimulation and EMG biofeedback provided five times a week for an average of 14 sessions. Each session consisted of 15 minutes of audiostimulation, 15 minutes of EMG biofeedback, and 15 minutes of simultaneous audiostimulation and biofeedback. Patients were asked to spend one half hour daily practicing the relaxation exercises used in the sessions. According to clinical assessments carried out before, during, and after the treatment, there was a gradual decrease in symptoms over the treatment period, accompanied by a concomitant reduction in resting EMG levels. Manns *et al.* concluded that EMG biofeedback and audiostimulation are effective treatment modalities in the myofascial pain-dysfunction syndrome.

The majority of biofeedback applications in the treatment of TMJ dysfunction have used EMG, although there is one report of Galvanic Skin Response (GSR) as the chosen modality. Gross (1975) used a combination of "Jaconson's" (sic) progressive relaxation and GSR feedback to train twenty MPD patients in general relaxation and anxiety reduction. Four months after the initial four-week training period involving one session per week, Gross reported a 90% improvement in function and symptoms. As well, the patients were notably less anxious and dealt better with stress.

In an interesting article describing the outcome of combining biofeedback and cognitive-behavioral psychotherapy, Stenn, Mothersill, and Brooke (1979), provided 11 MPD patients with eight weekly half-hour sessions of progressive relaxation; six of these were given auditory feedback from the masseter throughout the relaxation period; the remaining five were simply monitored for masseter muscle tension. Following the relaxation period, all the subjects met individually with a psychologist for cognitive therapy sessions focusing on the patient's pain response. Over the course of the treatment period, both EMG and pain levels dropped; however, only the subjects receiving feedback showed a significant reduction in pain, whereas there was no significant difference between groups in terms of EMG levels. The authors concluded that the reduction in pain could not be entirely due to the decreased masseter muscle tension, but must somehow be linked to the greater perception of control and competence resulting from the biofeedback training.

Not many studies have appeared which compare biofeedback therapy with other forms of conservative treatment for TMJ dysfunction. Dahlstrom, Carlsson, and Carlsson (1982) reported the results of biofeedback and occlusal splint therapy in a group of 30 female mandibular dysfunction patients. Fifteen of these subjects received up to six half-hour biofeedback sessions, the other 15 used a full coverage occlusal splint for a six week period. One month after the completion of the study the patients were reassessed and both groups showed significant improvement in clinical signs and symptoms although there was no significant difference between groups. The authors concluded that biofeedback and splint therapy are equally effective treatment modalities for mandibular dysfunction pain.

In another recent study comparing the effectiveness of biofeedback-assisted relaxation training, Brooke and Stenn (1983) found that relaxation training appeared to be as effective as other forms of conservative MPD treatment. No benefit was apparent when biofeedback was added to relaxation, nor did there appear to be a significant difference in long-term treatment outcome when the relaxation and biofeedback-relaxation patients were compared. On the other hand, in comparison with ultrasound and occlusal splint therapy, both relaxation and biofeedback-assisted relaxation therapy were more effective at the six month follow-up. When ultrasound and splint therapy were combined with relaxation training, there was an equalization of treatment outcome, but no significant difference between the various groups. Moss, Wedding, and Sanders (1983) also reported similar results in a study comparing relaxation training and masseter EMG feedback.

C. Summary

Masticatory muscle hyperactivity related to individual response to psychophysiological stress has been implicated as a primary etiological factor in the onset and maintenance of TMJ pain-dysfunction syndrome. From this theoretical position, a number of psychological, behaviorally oriented therapies have been proposed, the most common being progressive relaxation and biofeedback training. The application of these treatment modalities are based on the belief that reduction of overall muscle tension, especially masticatory muscle tension, will result in a decrease of pain. Additionally, training in relaxation and biofeedback techniques will improve patients' ability to cope with daily stress, thus weakening the cycle of the pain-dysfunction syndrome.

Although the results of biofeedback training in the syndrome appear positive and promising, most of the research to date has used small sample sizes or single case designs in evaluating the efficacy of the biofeedback. Larger sample sizes are needed to generate more conclusive findings. Even fewer studies have attempted to compare TMJ and non-TMJ patients on the basis of physiological data such as EMG activity--the assumption of significant differences between the two groups would seem to be an essential prerequisite for justifying biofeedback therapy. If significant physiological differences between the groups can be noted, it might be possible to determine the parameters within which biofeedback therapy would be indicated and provide the best results.

D. Research Questions

The present investigation was conducted to answer the following questions:

1. Is it possible to distinguish between TMJ dysfunction and non-TMJ dysfunction subjects on the basis of masseter and temporalis EMG levels?

2. Do the two groups respond differently to experimental stress?

3. What is the correlation between the number of positive self-report symptoms of mandibular dysfunction and EMG activity?

4. What is the correlation between the number of observed clinical signs of TMJ dysfunction and EMG activity?

III. METHOD

A. Subjects

The subjects for this study were selected from female volunteers between the ages of 18 and 49 who responded to a short anouncement circulated in a number of dentistry, dental hygiene, physical education, and educational psychology classes at the University of Alberta. The announcement briefly described the nature of the study and gave a short list of some of the symptoms commonly associated with TMJ dysfunction syndrome. Provision was made for volunteers who had positive TMJ symptoms as well as those who were asymptomatic but were interested in the study and wanted to participate. The former group consituted the "TMJ" group while the latter formed the "ASYMPTOMATIC" group.

From the approximately 45 respondents, some 30 reported TMJ symptoms and were admitted into the second phase of the screening procedure which included completing a more complete symptom checklist which had been adapted from a standard dental questionnaire (see Appendix C). To continue in the study each potential TMJ subject had to score "yes" to at least items 8 or 9, that is, the presence of pain in the temporomandibular joint or the sides of the face, or clicking of the joint, and not have undergone previous dental treatment for TMJ dysfunction. Following the positive questionnaire, each subject was then given a complete physical evaluation by a physiotherapist to assess the severity of the symptoms and suitability for inclusion in the study. All the subjects who possessed definite TMJ symptoms at this point were admitted to the experiment. A point of note, however, is that a number of subjects who manifested TMJ clicking or popping, or who may in the past have experienced facial pain, were admitted to the study as potential or borderline cases of TMJ dysfunction, even though they were not presently experiencing pain.

The selection of the ASYMPTOMATIC subjects proceeded in a similar manner except that none could have any positive TMJ symptoms or respond affirmatively to items 8 and 9 on the questionnaire. Some of the ASYMPTOMATIC subjects were also given a physical evaluation by the physiotherapist but most were accepted directly into the experiment. In selecting candidates for the ASYMPTOMATIC group, it should be noted that no assessment was made of physical factors other than those related directly to the temporomandibular joints and associated structures. While other types of physical dysfunction such as limited range of motion in the neck and back, and postural abnormalities may have been shared in common with the TMJ group, these variables where not specifically controlled for in the screening procedure. As a result, the distinction between the two groups may possibly have not been as precise as originally intended. It was recognized that because of the great diversity of secondary signs and symptoms involved in the TMJ syndrome, adequate control of all variables could not be possible and only primary signs of mandibular dysfunction could be considered.

In sum, then, for the purposes of the present study, the TMJ group was composed of subjects reporting and manifesting primary signs and symptoms of the TMJ syndrome; the ASYMPTOMATIC group consisted of individuals apparently free of mandibular dysfunction. In total, there were 24 ASYMPTOMATIC subjects and 30 TMJ subjects who participated in the study. Eventually, 6 of the TMJ group withdrew or were excluded because of incomplete data or unsuitability.

Within two to six weeks following the experiment, the TMJ subjects were asked to participate in a dental checkup in which the dentist evaluated each individual's occlusion and TMJ symptoms. This assessment included joint noise as well as muscle tenderness and pain upon palpation. The information gathered during the checkup was coded and recorded both specifically for individual muscles and collectively in terms of a cumulative TMJ pain score.

B. Research Design

The experiment was conducted along a 9 (conditions) by 2 (groups) repeated measures across condition phases format. The single-session experiment was conducted from mid-October to the end of December 1983.

Each subject's mean EMG levels, in microvolts, were independently recorded from the right and left temporalis and masseter muscles 36 times throughout the nine steps of the experiment. From these mean values, the maximum, minimum, and average EMG levels, as well as the standard deviation, were automatically calculated for each of the four muscles monitored at each step in the experiment. Further to this, the minimum level for each muscle was subtracted from its maximum value to render a difference score which was also included in the data pool. At this point, then, each subject's reaction to the experiment was represented by five separate measures of EMG activity for each muscle recorded over the nine steps in the experiment.

The EMG data collected were analyzed by means of a Hotelling's t^2-test for significant differences of MX, MN, AV, and DIFF between the two groups.[1] Once the preliminary analysis had been completed, further data were generated by calculating the sum of the means and the sum of the differences for each subject across the entire experiment. The resulting data were again tested for significant difference between groups with the Hotelling's t^2-test.

As the second step of the statistical analysis, Pearson product correlations were calculated between the EMG data and the TMJ symptom questionnaire. Finally, the EMG data were also correlated with the occlusal and pain scores gathered during the dental examination.

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[1] MX, MN, and AV refer respectively to the Maximum, Minimum, and Average EMG levels recorded by the apparatus during any single experimental time period. DIFF, the difference between the MX and MN values was subsequently calculated to provide a measure of the range of EMG activity for each step of the experiment.

C. Apparatus and Facilities

The EMG data were collected by a Biofeedback Research Institute Biocomp 2001 biofeedback system and stored on 5.25 inch floppy diskettes by the accompanying Apple micro-computer. Four sets of silver silver-chloride electrodes were used, one for each muscle group. Before application the skin was cleaned with an alcohol swab and then slightly abraded. The masseter electrodes were placed according to the standard placement described by Lippold (1967), with the two active electrodes over the belly of the muscle; one about 15 millimetres forward of the external acoustic meatus and the second about 15 millimetres diagonally anterior to the angle of the jaw. The reference electrodes were placed on the clavicle. For the anterior temporalis muscle the electrodes were placed level with the eyebrow, as close to the hairline as possible. Again, the reference electrodes were placed on the clavicle.

The experiment took place in relatively calm and quiet area in a physiotherapy clinic. The subjects were seated upright in a comfortable chair with their feet flat on the floor. In order to minimize EMG artifact, they were instructed to find a comfortable posture, keep their eyes closed, and refrain from extraneous movement during the data collection periods. In between the various steps of the experiment the subjects were allowed to open their eyes but they could not move from the chair. The entire experiment was of 45-60 minutes duration.

Experiment Procedure

The experiment consisted of a series of five stressors, each followed by a recovery period and prefaced by a general introductory briefing and an eight minute resting baseline with eyes closed. The first stressor consisted of a standard two minute mental arithmetic exercise involving the rapid serial subtraction of 7 from 1000. Subjects were told to proceed as quickly as possible, silently and without making any mistakes as an answer would be demanded when the time period was expired. After giving the answer, subjects were told to simply continue relaxing for a two minute recovery period. This type of mental activity is generally considered an effective psychological stressor which results in increased physiological arousal (Lorens & Darrow, 1962).

For the second stressor the subjects were to extend their legs and squeeze them together as hard as possible. The activity was maintained for one minute and followed by an equivalent recovery period. This stressor was adapted from Kydd (1959) who found that irrelevant masseter and temporalis muscle activity increased dramatically in TMJ subjects but not normal controls who were asked to exert maximum force with a foot against a stationary horizontal bar.

The third stressor consisted of a three minute soundtrack excerpt "Escape to Darkness" from the movie *Invasion of the Bodysnatchers* (Zeitlin, 1979), chosen for its emotionally arousing qualities. This was also succeeded by an equivalent recovery period.

The final steps in the experiment involved letting a cracker dissolve on the tongue for 3 minutes without masticating it; and again, an equal recovery period. This stressor was designed to elicit conditioned or proprioceptive responses resulting in heightened masticatory muscle activity. For the last stressor the subject was given another cracker which she was to chew thoroughly, swallow, and return to relaxing. The aim here was to record the pattern of EMG activity during a simple mastication exercise. No auditory feedback was provided during any part of the experiment and the subjects were seated facing away from the video monitor to eliminate any possible visual feedback. Following the completion of the experimental procedure, subjects were given a general synopsis of the findings and shown some of the graphs if interested. A full account of the experiment procedure, time periods and verbal instructions is offered in Appendix D.

IV. RESULTS

The analysis of data will be presented under four different groupings:

A. Subject characteristics as determined from the original screening questionnaire.

B. Occlusal and muscle palpation data derived from the clinical examination of the TMJ group.

C. EMG data collected during the experiment. The data is analyzed to reveal the differences in EMG activity between the two groups.

D. Correlations between:

i. EMG activity and questionnaire responses.

ii. EMG activity and the noise and palpation pain scores arising out of the clincial examination of the TMJ group.

A. Subject Characteristics

Table 1 indicates the characteristics of the subject population that was obtained through the screening questionnaire. The two key questions: pain in the jaw joint and/or face and, joint sounds and limitation of movement (8 and 9), which were used to assign subjects to the ASYMPTOMATIC or TMJ groups, appear to have been quite reliable in discriminating between the groups. Pain in the TM-joint or the face was present in 79% of the TMJ subjects, as compared to 17% and 9% of the ASYMPTOMATIC group. while joint sounds and limitation were reported by 75% of the group. Other items related to muscle pain and clenching or

Table 1

Screening Questionnaire

Characteristics of study population (N=48)

	Mean Age	Range
TMJ Group (N=24)	28.67	20-47
ASYMPTOMATIC Group (N=24)	26.42	18-39

		TMJ % Yes	ASYMPTOMATIC % Yes
1. Do you clench or grind your teeth during the day?		45	9
2. Have you been made aware of clenching or grinding your teeth during the night?		54	21
3. Do you have chronic headaches, or neck and shoulder pains?		54	21
4. Do you frequently have gastro-intestinal disturbances?		37	21
5. Do you ever wake up with an awareness of, or about, your teeth or jaw like you had them clenched in your sleep?		58	9
6. Do you have any awareness of the muscles of your neck or shoulders?		87	63
7. Do you have a tight or stiff neck?		70	34
8. Do you now, or have you ever had, pain in your jaw joint or the sides of your face (in and about the ears)?		79	17
9. Do you have a clicking jaw joint or have you ever experienced an inability to move your jaw or open your mouth widely?		75	9
10. Which side of your mouth do you chew on?	Both	45	66
11. Do you tend to breathe mostly			

through your nose?	83	87
12. Are you aware of persistent ringing in your ears?	16	5
13. Have you ever experienced pain or burning sensations in your:		
a. neck	54	46
b. shoulders	70	46
c. back	37	50
d. hips	20	21
14. Have you ever had treatment for problems of your:		
a. neck	25	21
b. shoulders	33	9
c. back	37	50
d. hips	20	5
15. Have you ever been told you have		
a. scoliosis	8	9
b. lordosis (swayback)	20	5
16. Have you ever been told you have a leg shortening on one side?	29	13
17. Have you ever suspected a leg shortening on one side?	12	13

bruxing also were good indicators of group membership. Diurnal clenching or bruxing was rated at 45% with nocturnal grinding and morning awareness of bruxing including 54% and 58%, respectively, of the TMJ sample. Conversely, only 9% of the ASYMPTOMATIC subjects reported clenching during the day or waking with and awareness of having clenched at night; 21% of this group reported having had their attention drawn to nocturnal bruxing. Headaches, neck and shoulder pains were reported by 54% and 70% of the TMJ group complained of a tight or stiff neck as contrasted with the ASYMPTOMATIC subjects of whom 21% and 34%, respectively, responded positively to these items.

The TMJ group varied in age from 20 to 47 with a mean age of 28.67 years, as compared to the ASYMPTOMATIC subjects who were between 18 and 39, rendering a mean age of 26.42 years. Although there was not a great age difference between the two experimental groups, the TMJ subjects were clearly more symptomatic overall. On the basis of the clinical examination, stress as a contributing factor to the symptoms was assessed in 84% of the TMJ subjects, and 74% of the group had never sought treatment for their symptoms. The assessment of stress in the TMJ subjects was a subjective rating on the part of the examining dentist, drawing from the subject's history, syptomatology, and self-report, as well as overall clinical impression. No other stress scales or rating procedures were used in this evaluation.

B. Occlusal and Muscle Palpation Data

Clinical examinations were arranged for nineteen of the TMJ subjects who agreed to continue with the study. The findings can be classified into three categories:

i. occlusal characteristics;
ii. joint sounds;
iii. palpation scores.

Occlusal Characteristics

Over half of the subjects (58-68%) possessed Class I occlusion according to the Angle classification system, the balance (31-36%) fell into Class II. Crossbite was observed in 15.8% of the sample, overbite of 2.5-4.5mm in 55.6%, and overjet of 1.5-3.0mm in 52.7% of the subjects. Nearly 79% of the group demonstrated anterior tooth contact, the range of maximum opening was 34-57mm, with 42.1% of the subjects reporting pain upon maximum opening. A more detailed analysis of the clinical data is presented in Table 2.

Joint Sounds

The examination registered sounds for each TMJ, and at various stages in the jaw movement cycle. A four-point rating scale was used to assess the severity of the signs: 0=absence of signs, 1=mild, 2=moderate, 3=severe. Overall, 84.2% of the subjects manifested some type of joint sound. Popping or clicking sounds upon mandibular movement were present in 63%, soft tissue grating in 26%, and hard tissue grating in only one individual (5.3%). No radiographs or other diagnostic procedures were carried out to further evaluate the nature of the observed joint sounds. A summary of joint sounds is presented in Table 3.

Each subject's joint noise scores were subsequently summed to provide an overall noise rating index to be used for comparing within the group. This noise index is reproduced in Table 4.

Table 2

Occlusal Data

	Angle Classification (% of sample N=19) I	II	III
Molar (R)	63.2	36.8	00.0
Molar (L)	68.4	31.6	00.0
Canine (R)	68.4	26.3	5.3
Canine (L)	57.9	36.8	5.3

Other characteristics		Percent of sample
Overbite	0.0-2.0mm	27.9
	2.1-4.5mm	55.6
	4.6-8.0mm	16.8
Overjet	0.0-1.5mm	31.7
	1.6-3.0mm	52.7
	3.1-7.0mm	15.9
Crossbite	(Yes)	15.8
Anterior contact	(Yes)	78.9
Painful to open to maximum	(Yes)	42.1
Range of maximum opening 34-57mm		

Palpation Scores

Temporomandibular Joint

In the clinical examination the TM-joints were palpated both laterally and posteriorly, with and without movement. The same four-point rating scale was used to assess degrees of pain or tenderness. Upon lateral palpation, 20% of the subjects reported pain.

Table 3

Joint Sounds

Location and type of sound	Severity	Percent of sample
Popping L (open)	1	31.6
	2	15.8
Popping R (open)	1	31.6
Soft tissue grating L (open)	1	15.8
Soft tissue grating R (open)	1	10.6
Hard tissue grating L/R (open)		0.0
Popping L (close)	1	26.3
Popping R (close)	1	10.5
Soft tissue grating L (close)	1	15.8
Soft tissue grating R (close)		0.0
Hard tissue grating L (close)		0.0
Hard tissue grating R (close)	1	5.3

Posterior palpation found tenderness in 53%. Detailed palpation results are documented in Table 5.

Muscles

Muscle tenderness was also evaluated on the four-point scale. The muscles were individually palpated and assigned scores which are reported in detail in Table 6. Overall, the most frequently painful muscle was the lateral pterygoid which was tender in 63% of the subjects. This was followed by the anterior temporalis

Table 4

Noise Scores (TMJ)

Cumulative Score	Percent of Sample
0	15.8
1	31.6
2	15.8
3	15.8
4	10.5
5	10.5

(58%), and the muscles of the suboccipital area (57.9%). The temporal tendon was also quite sensitive in the sample (47.4%) as was the deep masseter (42%). Sharav, Tzukert, and Rafaeli (1978) report similar incidences of muscle tenderness.

As with the noise scores, the palpation scores were also summed to produce a palpation tenderness index for the group. The results of this calculation are found in Table 7. Nearly half of the subjects (42.1%) obtained palpation sum scores between 10 and 19.

C. EMG Data Collected During the Experiment

During the experiment, temporalis and masseter muscle activity was monitored individually and bi-laterally for each step in the experiment. Each muscle occupied a specific

Table 5

Palpation Scores TM-Joint

	Severity	Percent of sample	Cumulative percent
TMJ laterally without movement	2	10.5	10.5
TMJ posterior without movement (L)	1	10.5	
	2	5.3	15.8
TMJ posterior without movement (R)	1	26.3	
	2	5.3	31.6
TMJ laterally with movement	1	5.3	
	2	5.3	10.6
TMJ posterior with movement (L)	1	10.5	
	2	5.3	15.8
TMJ posterior with movement	1	31.6	
	2	10.5	42.1

channel of the recording apparatus. In addition to an average EMG level calculated for each of the four muscles over each of the nine experimental conditions, the recording apparatus also generated values for maximum, minimum and standard deviation corresponding to each muscle. Over the course of the experiment 36 averages, maximum and minimum readings, and standard deviations were calculated for each subject. In answer to research question one, the preliminary analysis revealed there were no significant differences in EMG activity between the groups for any of the individual muscles throughout the nine experimental conditions. With a view to facilitating the analysis and presentation of this

Table 6

Muscle Palpation Scores

Muscle	Severity	Percent of sample	Cumulative percent
Post. temporalis(L)	1	10.5	10.5
Post. temporalis (R)	1	21.1	21.1
Ant. temporalis (L)	1	31.6	
	2	21.1	52.7
Ant. temporalis (R)	1	31.6	
	2	26.3	57.9
Superficial masseter (L)	1	15.8	15.8
Superficial masseter (R)	1	10.5	10.5
Deep masseter (L)	1	10.5	
	2	15.8	
	3	5.3	31.6
Deep masseter (R)	1	15.8	
	2	21.1	
	3	5.3	42.2
Hyoid, ant. digastric (L/R)	0	100.0	100.0
Post. digastric (L)	1	10.5	10.5
Post. digastric (R)	1	15.8	
	2	5.3	21.1
Sternomastoid (L)	1	26.3	
	2	5.3	31.6
Sternomastoid (R)	1	26.3	
	2	10.5	36.8
Suboccipital area (L)	1	26.3	
	2	26.3	
	3	5.3	57.9
Suboccipital area (R)	1	26.3	
	2	26.3	
	3	5.3	57.9
Medial pterygoid (L)	1	10.5	
	2	15.8	26.3

Medial pterygoid (R)	1	5.3	
	2	15.8	21.1
Lateral pterygoid (L)	1	26.3	
	2	42.1	68.4
Lateral pterygoid (R)	1	31.6	
	2	26.3	
	3	5.3	63.2
Temporal tendon (L)	1	26.3	
	2	15.8	
	3	5.3	47.4
Temporal tendon (R)	1	26.3	
	2	15.8	
	3	5.3	47.4
Stress implicated as a contributing factor		(Yes)	84.2
Previous treatment for TMJ problem		(No)	73.7

large amount of information, the four averages collected were summed to produce a total EMG score for each subject at every phase in the experiment.

The sum of the averages for both groups were calculated and tested for significant differences between groups by means of a Hotelling 2-sample t^2-test, the outcome of which is presented in Table 8. A review of the results shows that the TMJ group produced higher levels of EMG activity than the ASYMPTOMATIC group in all of the experimental conditions excluding step 2, the mental subtraction exercise in which there was very little distinction between the performance of both groups. The greatest difference in EMG activity between the two groups occurred during steps 4 and 8 of the

Table 7

Muscle Palpation Cumulative Scores

Cumulative Score	Percent of Sample
0-9	36.8
10-19	42.1
20-30	21.1

experimental procedure, the physical exertion and cracker dissolving stressors. In these tests the ASYMPTOMATIC group produced 10.080 and 9.961 EMG microvolts respectively, compared to 14.217 and 13.764μv in the TMJ group. Although the difference between the two groups is of 4.137μv for condition 4 and 3.803μv for condition 8, the difference is not significant at the .05 confidence level ($p > .05$) due to the large standard deviation. The conclusions are the same for every step of the experiment; while there are differences in absolute magnitude between the two groups, the large standard deviations preclude statistical significance.

In a second analysis, again in response to the first research question, the minimum EMG levels were subtracted from the maximum EMG levels for each channel to generate a difference score for each channel. The four derived difference scores were then summed and subjected to the same Hotelling t^2-test described above. The results are presented

Table 8

Sum of channel means for each experimental condition (EMG microvolts)

	ASYMPTOMATIC GROUP		TMJ GROUP	
Condition	Mean	SD	Mean	SD
Baseline	8.343	2.748	9.228	4.060
Serial subtraction	11.997	8.009	11.549	4.982
Recovery	9.550	4.401	10.650	6.777
Physical exertion	10.080	4.970	14.217	15.420
Recovery	8.731	3.373	10.340	6.861
Soundtrack	9.080	3.573	10.492	4.708
Recovery	7.405	2.510	9.688	6.752
Cracker dissolving	9.961	4.341	13.764	8.052
Cracker mastication	24.037	7.112	25.062	6.143

Table 8 (Part 2)

Tests for each variable with
multivariate degrees of freedom

Var	t^2	*df1*	*df2*	*F*	*P*
1	0.783	9	38	0.072	1.000
2	0.054	9	38	0.005	1.000
3	0.444	9	38	0.041	1.000
4	1.565	9	38	0.144	0.998
5	1.062	9	38	0.098	1.000
6	1.370	9	38	0.126	0.999
7	2.411	9	38	0.221	0.990
8	4.148	9	38	0.381	0.937
9	0.286	9	38	0.026	1.000

in Table 9 and show that, again, the TMJ group produced a consistently greater range of EMG activity over all the experimental conditions compared to the ASYMPTOMATIC group. Again, however, there was no statistically significant difference between the two groups.

On the whole, the TMJ group appeared more reactive to the various experimental stressors than did the ASYMPTOMATIC group, as evidenced by the deviations from the baseline mean EMG microvolt level for each experimental condition. The mean deviation from baseline for conditions 2 to 8 was $2.300\mu V$ in the TMJ group as compared with $1.467\mu V$ in the ASYMPTOMATIC group. The greatest within group deviations

Table 9

Sum of the difference between maximum and minimum EMG levels for each experimental condition

	ASYMPTOMATIC GROUP		TMJ GROUP	
Condition	Mean EMG level (microvolts)	SD	Mean EMG level (microvolts)	SD
Baseline	7.702	4.796	9.511	9.980
Serial subtraction	14.525	12.652	17.077	19.417
Recovery	11.462	6.557	17.240	23.355
Physical exertion	11.793	10.897	24.003	30.270
Recovery	14.420	15.738	20.231	22.811
Soundtrack	13.204	15.026	21.183	18.508
Recovery	8.541	5.638	14.696	18.873
Cracker dissolving	15.389	9.045	23.729	25.847
Cracker mastication	114.746	22.603	115.791	26.962

Table 9 (Part 2)

Tests for each variable with
multivariate degrees of freedom

Var	t^2	*df1*	*df2*	*F*	*P*
1	0.641	9	38	0.059	1.000
2	0.291	9	38	0.027	1.000
3	1.361	9	38	0.125	0.999
4	3.455	9	38	0.317	0.964
5	1.055	9	38	0.097	1.000
6	2.688	9	38	0.247	0.985
7	2.343	9	38	0.215	0.991
8	2.226	9	38	0.204	0.992
9	0.021	9	38	0.002	1.000

from baseline occurred during the mental subtraction stressor (3.654μV) for the ASYMPTOMATIC group and, the physical exertion and cracker dissolving conditions (4.989, 4.536 microvolts respectively) for the TMJ group.

In response to research question two, analyses of variance were conducted between baseline and the seven subsequent experimental conditions. Statistical significance at the .001 level was found for the serial subtraction and cracker dissolving conditions. The soundtrack and physical exertion stressors were also significant at the .01 and .05 levels respectively. For the recovery periods, only condition three, recovery from the serial subtraction, produced a significant difference from the baseline ($p < .01$). The recovery period following the physical exertion condition was nearly significant at the .05 level, while the soundtrack recovery was not. Although all but one of the experimental conditions, produced a significant variation from baseline for all of the experimental subjects, only the cracker dissolving stressor approached significance ($p < .05$) in distinguishing between the groups. Summaries of the analyses of variance are presented in Appendix E.

D. Correlations

EMG and Questionnaire Responses

In terms of the third research question, an analysis of the sum of the average EMG levels for each channel and the screening questionnaire was conducted to determine the nature of the correlation between increases in positive questionnaire responses and EMG levels for each experimental condition. The Pearson product correlations were positive and significant for three of the experimental conditions: soundtrack ($r = .2433$, $p < .05$), cracker dissolving ($r = .2898$, $p < .05$) and cracker mastication ($r = .2433$, $p < .05$). Significance was also approached in condition 7, recovery from the soundtrack, and the total of the average EMG levels summed across the entire experiment. The correlations between the questionnaire responses and the mean EMG levels for each step of the experiment are presented in Table 10.

The same Pearson product correlation analysis was then carried out between the difference scores and the questionnaire results to determine the correlation between increasing range of EMG activity and increasing positive questionnaire responses. Unlike the analysis described above, no correlations were significant at the .05 level. Only one experimental condition, physical exertion, produced a correlation approaching significance. The results of this analysis are found in Table 11.

EMG Activity and the Noise and Palpation Pain Scores

In response to research question four, Pearson product correlation coefficients were calculated for the mean EMG level at each experimental condition and the cumulative noise and palpation pain scores for the TMJ subjects. Table 12 indicates that there were no significant correlations ($p < .05$) between EMG activity and TMJ noise scores. An additional analysis revealed there was no significant correlation between anterior tooth contact, degree of overbite and the noise scores in the sample.

On the other hand, the same analysis carried out between the average EMG levels and the palpation pain scores produced significant or near significant correlations for seven of the nine experimental conditions. The strongest correlation ($r = .5402$, $p < .01$) between rising EMG activity and muscle tenderness occurred in the second step of the

Table 10

Sum of mean EMG levels correlated with Questionnaire

Condition	*r*	*p*
Baseline	.1948	.092
Serial subtraction	.0537	.092
Recovery	.1137	.221
Physical exertion	.1851	.104
Recovery	.1663	.129
Soundtrack	.2511	.043
Recovery	.2233	.064
Cracker dissovling	.2898	.023
Cracker mastication	.2433	.048
Total sum for experiment	.2219	.065

experiment, the mental subtraction stressor. No significant correlations between EMG activity and muscle pain were noted for either the cracker dissolving or mastication conditions. As with the noise scores, correlation coefficients were obtained for the relationship between pain scores and anterior contact and degree of overbite. No significant correlation was noted between anterior contact and muscle pain, but a moderate correlation existed for degree of overbite and muscle tenderness (r=.4803, p <.05). The results of this analysis are detailed in Table 13.

Table 11

Sum of difference scores correlated with Questionnaire

Condition	*r*	*p*
Baseline	.1260	.197
Serial subtraction	.1036	.242
Recovery	.0006	.498
Physical exertion	.2113	.075
Recovery	.0394	.395
Soundtrack	.1801	.110
Recovery	.1226	.203
Cracker dissolving	.1709	.123
Cracker mastication	.1005	.248
Total sum for experiment	.1029	.234

E. Summary

The screening questionnaire completed by all the participants in the study appears to have been quite reliable in assigning group membership as evidenced by the clearcut differences in response between the two groups. Clinical examination of the majority of the TMJ subjects found signs of mandibular dysfunction which corroborated the self-reported symptoms. All of the experimental conditions proved effective in eliciting significant variation from baseline in the experimental subjects. However, statistical analysis of EMG activity throughout the nine experimental conditions, conducted on the basis of group membership, did

Table 12

Correlation of noise scores with mean EMG

Condition	*r*	*p*
Baseline	.0889	.359
Serial subtraction	-.1071	.331
Recovery	-.0002	.500
Physical exertion	.1144	.320
Recovery	.1377	.287
Soundtrack	.1491	.271
Recovery	.1134	.322
Cracker dissolving	.2605	.140
Cracker mastication	.1625	.253
Overbite	-.0987	.348

not, however, reveal any significant differences between the ASYMPTOMATIC and TMJ groups. Conversely, there were significant relationships between clinically determined levels of muscle tenderness and EMG activity measured during the various experimental conditions.

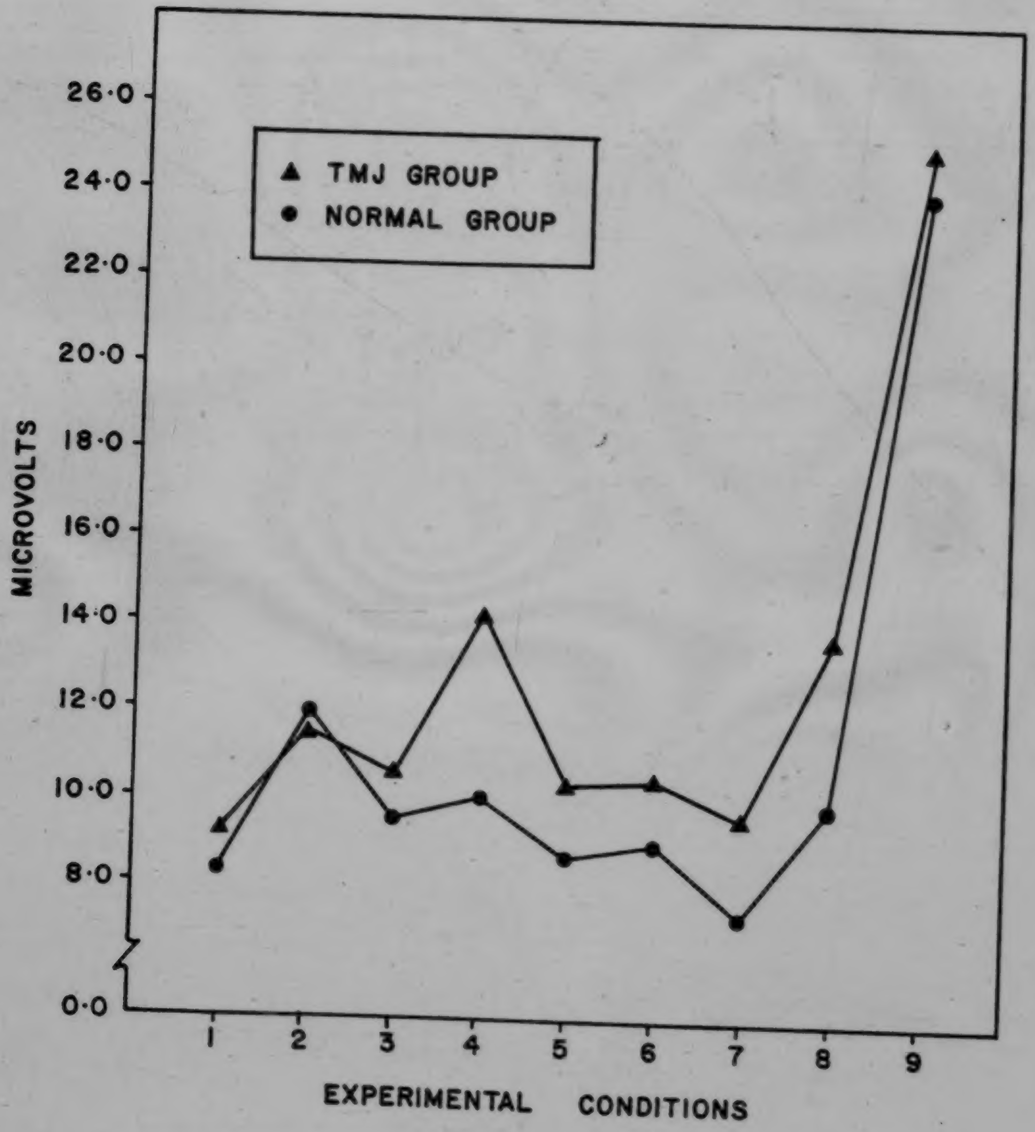
Table 13

Correlation of palpation scores with mean EMG

Condition	*r*	*p*
Baseline	.4200	.036
Serial subtraction	.5402	.008
Recovery	.4401	.029
Physical exertion	.4317	.032
Recovery	.3981	.045
Soundtrack	.3743	.057
Recovery	.4568	.024
Cracker dissolving	.2438	.157
Cracker mastication	.3020	.104
Overbite	.4803	.022

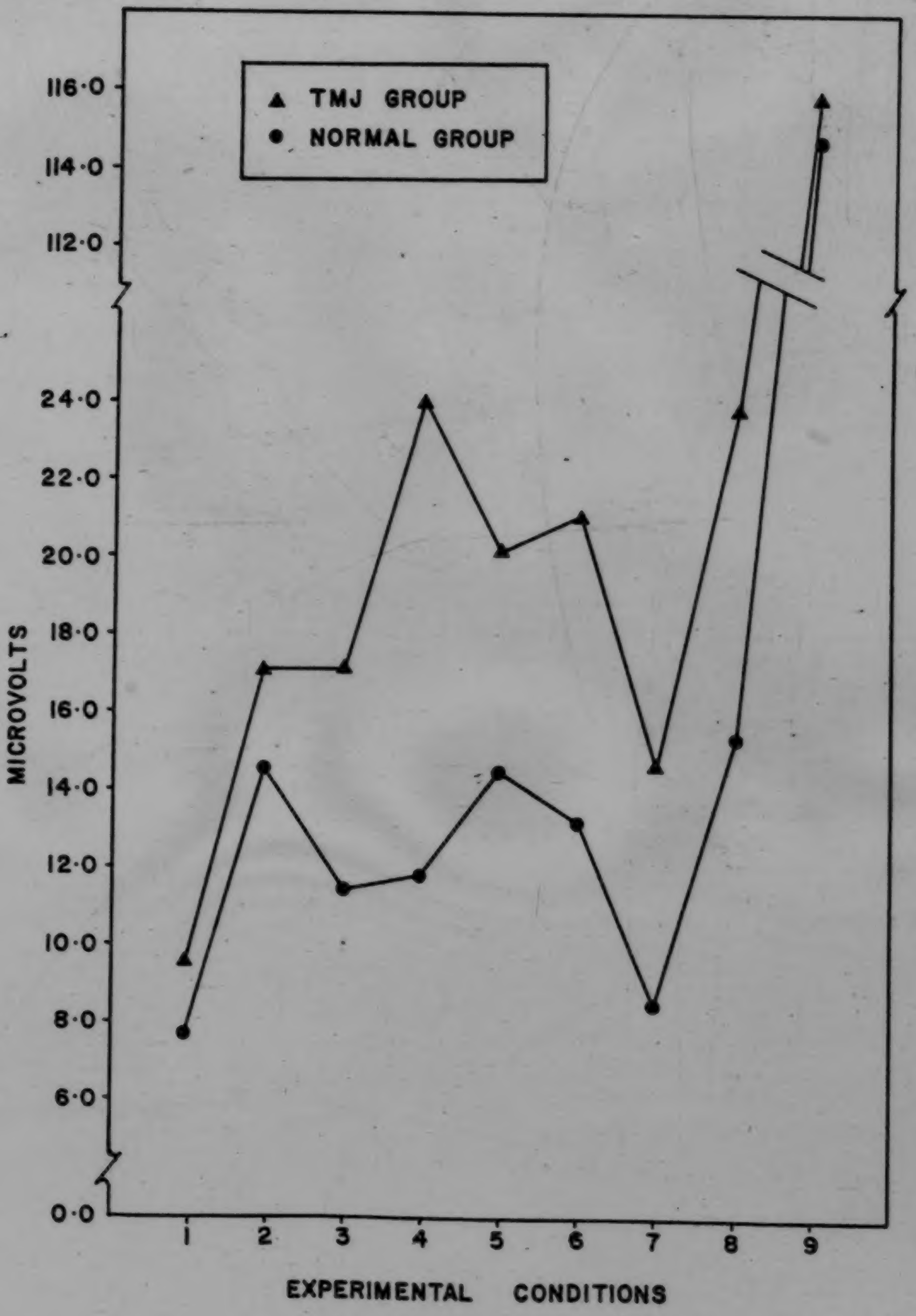
Sum of channel means for each experimental condition

FIGURE I



Sum of the difference between maximum and minimum EMG levels for each experimental condition

FIGURE 2



V. DISCUSSION

A. Major Findings

The results of the present study show that while the experimental conditions were effective in producing significant differences from baseline EMG levels in all the subjects, there were no significant differences in EMG activity between the TMJ and the ASYMPTOMATIC groups at baseline, nor during the course of the experiment. Although the mean EMG levels were generally greater in the TMJ group as compared with the ASYMPTOMATIC group, the differences between the groups were not significant at the .05 level. This is likely due to the large internal variablity of EMG levels observed in both groups. Thus, no firm distinctions could be drawn between the groups on the basis of EMG activity. Details of this analysis of variance are presented in Appendix E.

Some of the variability encountered in both experimental groups must be attributed to individual differences. On the other hand, the TMJ group exhibited generally greater variability than the ASYMPTOMATIC group; this would suggest some factor other than purely individual differences was operating. The analysis of the clinical findings, especially muscle and joint palpation scores, and the mean EMG levels, indicates that as the presence of the former increases, so does muscle tension as measured on the electromyograph. Consequently, the variability of EMG levels within the TMJ group may be linked to the number and severity of pain-dysfunction signs found during the clinical examination.

B. Limitations and Delimitations

In establishing the bounds of the present study, the primary criterion was the presence or absence of TMJ syndrome signs in the sample. That is, pain in or about the temporomandibular joint and clicking, popping, or grating sounds in the joint upon movement. Subjects reporting these signs were assigned to the TMJ group, while the others were placed in the ASYMPTOMATIC group. This rather specific delimitation, although seemingly precise, was also the source of a number of important limitations which may have contributed to the lack of significant difference observed between the two experimental groups.

One of the major limitations of the study was the inability to control for physical variables not measured or otherwise assessed by the screening questionnaire, the EMG apparatus, or direct physical examination. These uncontrolled variables may have obscured the distinction between the two experimental groups. Most obviously, only the TMJ group was given a clinical examination during which the dentist evaluated occlusal characteristics and palpated for muscle tenderness. If the ASYMPTOMATIC group had been subjected to the same type of assessment procedure, certain subjects might have been excluded from the study or reassigned to the TMJ group. Similarly, neither of the groups were examined for other than occlusal and masticatory muscle characteristics. This may have also imposed a serious limitation on the study given the potential impact of the TMJ syndrome upon the entire body (Gelb, 1977). A thorough physical examination of all subjects by the physiotherapist would undoubtedly have resulted in better defined groups.

There were also some limitations inherent in the structure of the experiment itself, the most notable being the measurement of masseter and temporalis activity with the mandible stationary in a rest position (excluding the mastication condition). The lack of difference in EMG levels between the two groups might not have occurred if muscle activity had been recorded during some type of mandibular movement cycle, for example, an open/close sequence where the mouth is opened to maximum and then returned to rest. With limitation of mandibular movement, due to spasm or overactivity of the elevator muscles (trismus), being one of the principal symptoms of the TMJ syndrome, one might expect to observe differences between symptomatic and asymptomatic subjects in terms of masseter and temporalis EMG activity throughout the movement cycle. In cases of trismus these muscles would oppose the jaw opening action of the depressor muscles and lead to elevated EMG levels in masseter and temporalis when the mouth is opened. It follows that monitoring masseter and temporalis activity through a complete open/close sequence might have revealed important differences between the ASYMPTOMATIC and TMJ groups.

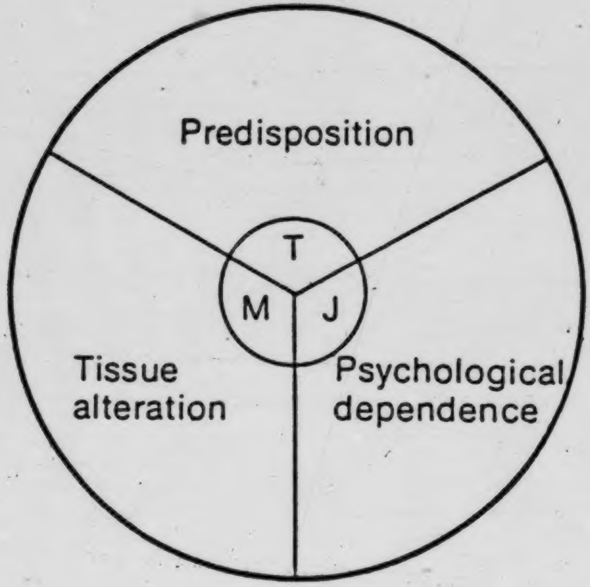
At this point it may be useful to make the distinction between subjects in the TMJ group who manifested some signs and symptoms of the TMJ pain-dysfunction syndrome but who, on the whole, were not experiencing pain at the time of the experiment. These participants could be termed pre or sub-clinical TMJ dysfunction patients. Since the screening questionnaire did not distinguish between past or present pain related to mandibular dysfunction, both types of subjects were included in the sample. More specific and careful screening would likely have selected a more uniform TMJ sample.

Another factor to consider is the role of stress as a contributory element in the onset and development of the TMJ pain-dysfunction syndrome. Gelb (1977) describes the syndrome in terms of a TMJ triad which includes: (a) predisposition, (b) tissue alteration and, (c) psychological dependence (pp. 1-31). According to Gelb, unless all three components of the triad are present and of sufficient magnitude, clinical signs and symptoms of the syndrome will not appear.

Although a stress component was assessed in 84% of the TMJ subjects, this rating was based only on the subject's self-report, history, and the clinical judgement of the examining dentist. Each subject's stress level might have been better evaluated through the use of some type of individually administered instrument such as the *Social*

Figure 3

TMJ triad (Gelb, 1977, p. 4)


Predisposition
T
M
J
Tissue alteration
Psychological dependence

Readjustment Rating Scale (Holmes & Rahe, 1967). From the perspective of Gelb's model (1977), it would appear that while all the TMJ subjects manifested some degree of mandibular dysfunction, only a limited number demonstrated the necessary combination of stress, predisposition, and tissue alteration leading to actual pain and other clinical signs and symptoms. With a more objective measure of stress at its disposal, the study could have added this criterion in the group selection procedure alongside the physical manifestation of TMJ dysfunction; this would also have contributed to better group definition and the likelihood of significant differences between groups. Finally, if an analysis had been carried out only on the data recorded from the subjects who were actually experiencing pain at the time of the study, significant differences between the two experimental groups might have been revealed.

C. The Research Questions

The present investigation was conducted to consider four specific questions related to the use of EMG biofeedback in the treatment of the temporomandibular joint pain-dysfunction syndrome. Each of these questions will now be addressed.

Question One: Is it possible to distinguish between TMJ dysfunction and non-TMJ dysfunction subjects on the basis of masseter and temporalis EMG levels?

From the analysis that was conducted on the average, maximum, and minimum EMG levels for each muscle monitored throughout the experiment, no significant differences between the two experimental groups were apparent. Further analysis of total mean EMG levels, and total difference in EMG activity per experimental condition also failed to find significant differences between the groups. As explained above, this lack of significant difference between groups is related to the large within groups EMG variability.

Negative results in the present study, however, do not preclude the possiblity of certain TMJ subjects, likely those manifesting the greatest severity of clinical signs and symptoms, showing significant differences from either their own group or the ASYMPTOMATIC group. Indeed, the EMG values recorded for the ASYMPTOMATIC subjects could serve as preliminary normative data to be used in determining the presence of abnormal muscle tension, and setting appropriate treatment goals for biofeedback therapy.

Question Two: Do the two groups respond differently to experimental stress?

An analysis of variance between the baseline condition and the following seven stressors and recovery periods concluded that the stressors produced significant variation from baseline. However, no significant difference was found in the way the TMJ and ASYMPTOMATIC groups responded to the experimental conditions. As mentioned previously, this finding may be due to a number of factors such as inadequate group definition and assignment prodedures and EMG monitoring with the mandible in a static, rest position only.

Question Three: What is the correlation between positive self-report symptoms of mandibular dysfunction and EMG activity?

Of the nine experimental conditions, three produced weak but significant correlations between mean EMG levels and the number of self-reported symptoms. There were no significant correlations between mean EMG difference scores and questionnaire responses. It may be concluded that, while subjective, self-report symptom checklists or questionnaires may be useful in screening respondents for potential mandibular dysfunction, these are not reliable predictors of muscle tension levels.

Question Four: What is the correlation between observed clinical signs of TMJ dysfunction and EMG activity?

The present study did not find a significant correlation between the severity of temporomandibular joint noise and EMG levels. Thus, the extent of joint dysfunction is not necessarily an indicator of possible muscle tension abnormalities. On the other hand, there was a significant relationship between the degree of overbite and mean EMG activity over the experimental conditions. This finding supports the clincal observation that malocclusion is often accompanied by abnormal muscle activity.

Significant correlations were also observed between muscle tenderness scores and mean EMG levels for seven of the nine experimental conditions. This would tend to confirm the position that increased pain is accompanied by increased muscle tension. Alleviation of pain may therefore follow reduction of the muscle tension as noted by Jacobson (1967). Biofeedback therapy should here be considered a viable treatment modality for managing the muscular pain associated with the TMJ pain-dysfunction syndrome.

D. Directions for Further Research

The present study has emphasized the need for careful and systematic differential diagnosis of the TMJ pain-dysfunction syndrome. Evidently, as research progresses, the range and interaction of the constitutive elements of the syndrome increase both in number and complexity, necessitating a thorough and individual assessment of each patient. Each examination should include an evaluation of immediately obvious factors such as occlusion, posture, recent trauma etc.; but also a complete history, physical and psychological, should be obtained. Biofeedback therapy is indicated as a potentially effective treatment in the pain-dysfunction syndrome--when the psychophysiological signs are present.

As in virtually all biofeedback/TMJ research, muscle tension as indicated by EMG levels was the only physiological modality measured. Here the results were inconclusive, and this may have been due to inadequate subject selection prodedures, and other factors mentioned previously. Further EMG research should attempt to screen for large clinical samples that manifest specific signs and symptoms. One can hope that more uniform sample populations will reduce the large within group EMG variability witnessed in the present study, and result in more definitive and significant conclusions.

In addition to more thorough screening procedures, the establishment of a psychophysiological assessment protocol for TMJ patients would be useful both diagnostically and in descriptive research. Such a protocol should include EMG measurements with the mandible at rest; immobile at various stages in the movement cycle; and in motion, charting the action of the muscle throughout an entire movement cycle. One might also add lateral, retrusive, and protrusive movements to the protocol. Different types of stress, pain, or illness behavior assessment instruments might also be included in the programme to provide a more complete picture from the physiological and psychological perspectives.

Another point to consider in further research is the type of analysis conducted on the data. In the present study, the EMG readings for each muscle were summed to provide a total muscle activity score for each subject. While this may have been useful in testing for significant differences in absolute magnitude between experimental groups, it obscured potentially significant muscle activity

6

patterns within individual subjects. Such patterns related to idiosyncrasies of orofacial balance and occlusion may ultimately be more important in in the onset and progression of the TMJ pain-dysfunction syndrome than indications of overall muscle activity. Further EMG studies should look at the relationship between masticatory muscle/occlusion equilibrium and TMJ dysfunction in individuals as well as in overall group comparisons.

Since EMG research is the most obvious choice in the TMJ pain-dysfunction syndrome, other biofeedback modalities have been generally neglected. However, in the light of the apparently important role of stress response and other psychological factors in the etiology and maintenance of the syndrome, further investigations into the psychophysiological response profiles of TMJ patients should be conducted. To date, Gross (1975) is the only worker to have reported successful treatment of a number of pain-dysfunction patients using only galvanic skin response (GSR) feedback and progressive relaxation. Additional research into the judicious application of the various psychophysiological modalities available to current biofeedback technology may provide the therapeutic answer to the patient who has been unresponsive to conventional therapies. Essential to the success of such an endeavour is, again, continued research in the area of response specificity. The unresponsive patient may represent the therapist's failure to identify and work with the dominant psychophysiological response pattern. Multi-modality stress response profiles of normal subjects and pain-dysfunction patients would be a first step in determining whether or not other types of biofeedback therapy are potentially useful in TMJ treatment.

The present study can be best identified with the psychophysiological theory of the TMJ pain-dysfunction syndrome. In holding with this theoretical framework, the use of biofeedback in the treatment of TMJ patients rests upon evidence of excessive masticatory muscle activity, most commonly resulting from stress. While the present study did not find significant differences between the two experimental groups, both did, however, react to the experimental conditions with significant increases in masseter and temporalis EMG activity--a finding which supports the relationship between stress and masticatory muscle hyperactivity. More refined selection and assessment procedures, as described above, would likely have added to the significance of the study.

In devising a treatment program for the TMJ pain-dysfunction patients from the the psychophysiological point of view, the role of stress and masticatory muscle hyperactivity in the emergence of the syndrome must be considered. As part of this theoretical framework, biofeedback and related psychological procedures are an obvious treatment option. However, in light of Gelb's TMJ triad (1977), a treatment protocol of biofeedback, psychotherapy, stress management, psychological assessment and management of pain and illness behavior accounts only for the psychological dependence element, and ignores the categories of predisposing factors and tissue alteration. Conventional dental and physical therapies are here indispensable.

The theoretical understanding of the temporomandibular joint pain-dysfunction syndrome has evolved from a purely mechanical perspective on etiology and treatment, to an elaborate view of the interaction of physiological and psychological influences. Treatment of the disorder has consequently assumed an increasingly multi-disciplinary nature. As the understanding and treatment of the syndrome continues to grow and develop, biofeedback and other forms of psychophysiological therapy will serve an important function within this complex therapeutic endeavor.

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Appendix A

Ankylosis: *a. bony b. fibrous. Bony ankylosis* the union of the bones of a joint by proliferation of bone cells, resulting in complete immobility. b. *Fibrous ankylosis* reduced mobility of a joint due to proliferation of fibrous tissue (Friel, 1974, p. 99). These are the most common forms of ankylosis found in the temporomandibular joint.

Articular Disc a pad of dense fibrous tissue that is nonvascular and noninnervated except in the peripheral areas, lying between the condyle of the mandible and the mandibular fossa. The disc divides the joint into two cavities and provides an articulating surface between the condyle and fossa. Also referred to as the **Meniscus** (Bell, 1982; Ermshar, 1982).

Bruxism rhythmic or spasmodic grinding of the teeth in other than chewing movements of the mandible, especially such movements performed during sleep. Dental malocclusion and tension-release factors are the usual inciting causes (Friel, p. 231).

Condyle the rounded projection on a bone, usually for articulation with another. Here, the articular surface of the mandible upon which rests the articular disc (Friel, p. 350; Hylander, 1980).

Crepitus scraping or grating sounds produced in the joint upon articulation. Often related to perforation of the articular disc resulting in bony contact.

Meniscus see Articular Disk

Muscle Spasm a sudden involuntary contraction of a muscle or group of muscles that are functionally related. It is attended by pain and interference with function, and it is manifested by involuntary rigidity, distortion, or movement. Clonic muscle spasm is of momentary duration; tonic spasm persists for a period of time. Cycling muscle spasm is protracted tonic spastic activity that becomes self-perpetuating, presumably as the result of pain incidental to continued spastic contraction of the muscle. Isometric spasm causes muscular rigidity with marked resistance to stretch; isotonic spasm causes shortening of the muscle, which produces distortion or skeletal movement (Bell, p. 57).

Muscle Splinting a protective mechanism whereby a threatened or injured component of the musculoskeletal system is immobilized by increased tonicity of its surrounding musculature. The condition usually subsides when the threat or injury disappears. Prolonged splinting may lead to muscle spasm (Bell, p. 57).

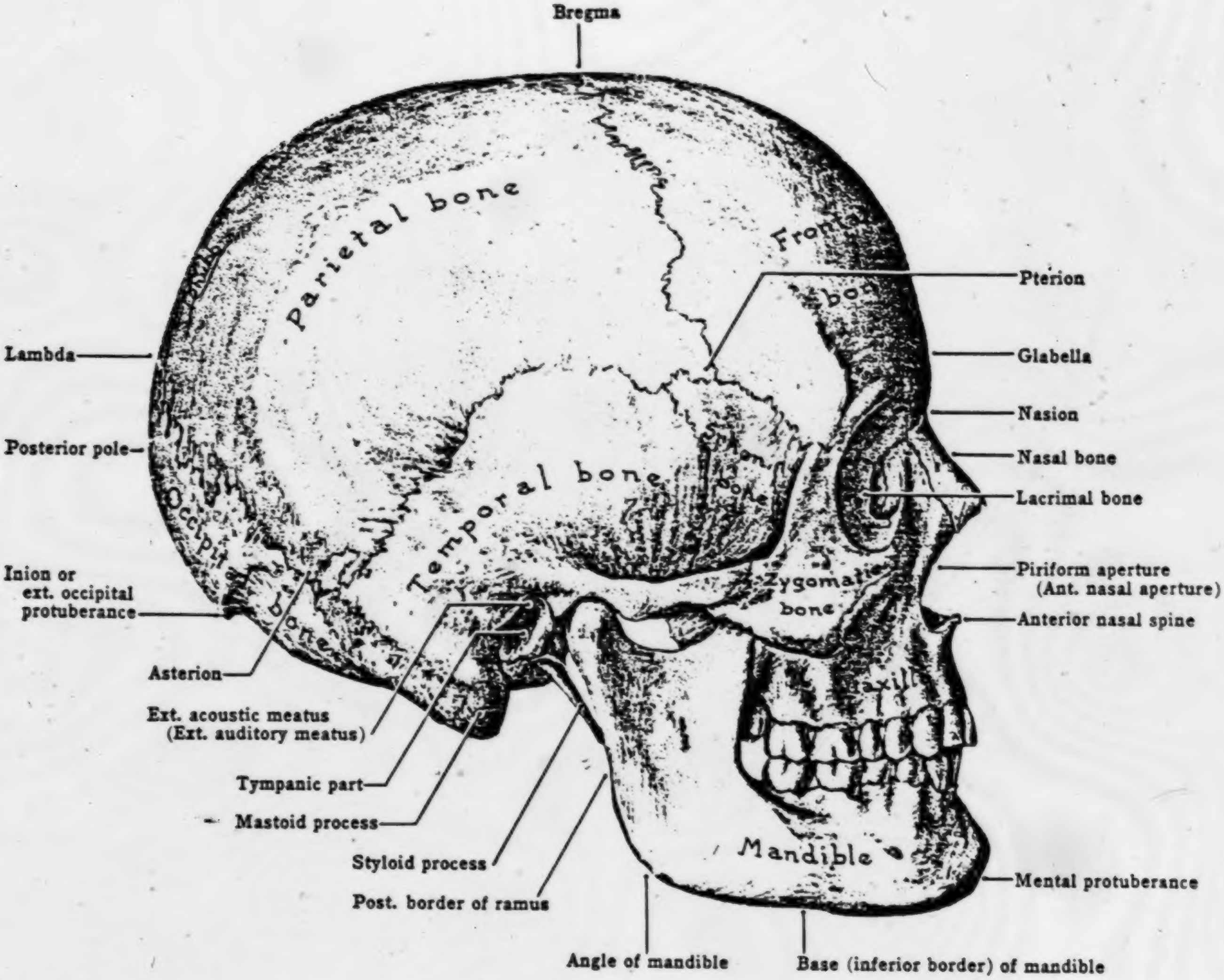
Muscle Tonus the resistance of the muscle to elongation or stretch. *Hypertonicity* refers to a relative increase in passive resistance to stretching the muscle; *hypotonicity* refers to a decreased passive resistance to stretch. Muscle tonus serves two purposes: (1) it furnishes the muscular activity needed to maintain sharp contact of the articulating parts in joints when at rest or under negative interarticular pressure imposed by the effect of gravity, and (2) it maintains the muscles in an optimum state of readiness for contraction (Bell, pp. 56-57).

Tinnitus a noise in the ears, as ringing, buzzing, roaring, clicking, etc. Such sounds may at times be heard by others than the patient (Friel, p. 1613).

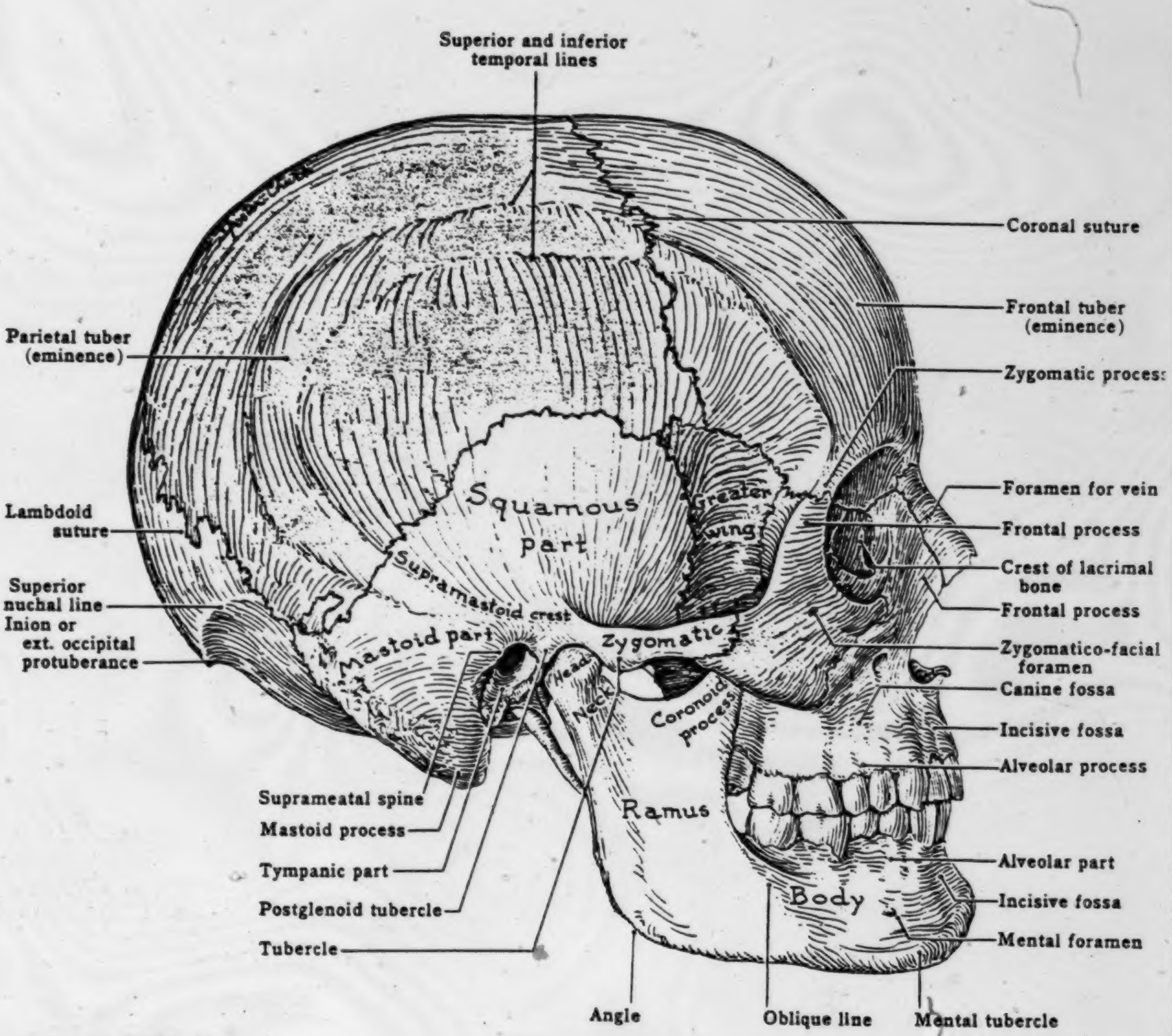
TMJ the temporomandibular joint, the jaw joint. Often used alone as an abbreviation for the temporomandibular joint pain-dysfunction syndrome.

Appendix B

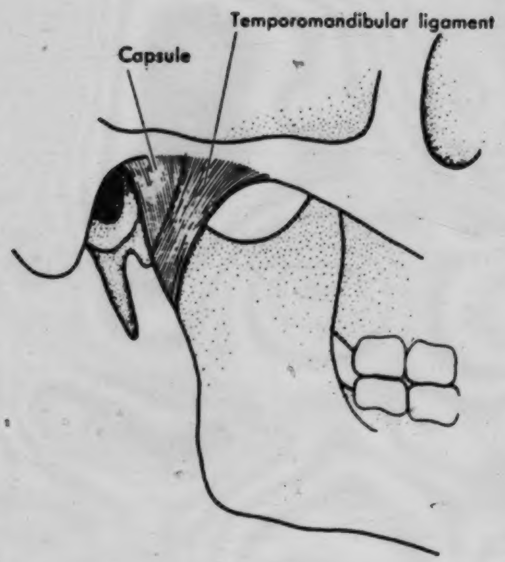
Anatomical figures


Bregma
Parietal bone
Frontal bone
Pterion
Glabella
Lambda
Nasion
Posterior pole
Nasal bone
Lacrimal bone
Temporal bone
Occipital bone
Piriform aperture
(Ant. nasal aperture)
Zygomatic bone
Inion or
ext. occipital
protuberance
Anterior nasal spine
Asterion
Ext. acoustic meatus
(Ext. auditory meatus)
Tympanic part
Mastoid process
Styloid process
Mandible
Post. border of ramus
Mental protuberance
Angle of mandible
Base (inferior border) of mandible

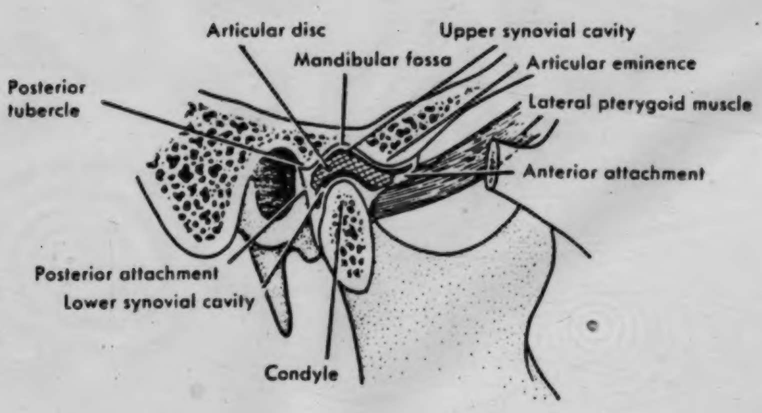
Lateral view of the skull (Anderson, 1978, p. 7-6).


Superior and inferior temporal lines
Coronal suture
Frontal tuber (eminence)
Zygomatic proces
Parietal tuber (eminence)
Foramen for vein
Frontal process
Crest of lacrimal bone
Frontal process
Lambdoid suture
Superior nuchal line
Inion or ext. occipital protuberance
Zygomatico-facial foramen
Canine fossa
Incisive fossa
Alveolar process
Squamous part
Greater wing
Supramastoid crest
Mastoid part
Zygomatic
Head
Neck
Coronoid process
Ramus
Body
Suprameatal spine
Mastoid process
Tympanic part
Postglenoid tubercle
Tubercle
Alveolar part
Incisive fossa
Mental foramen
Angle
Oblique line
Mental tubercle

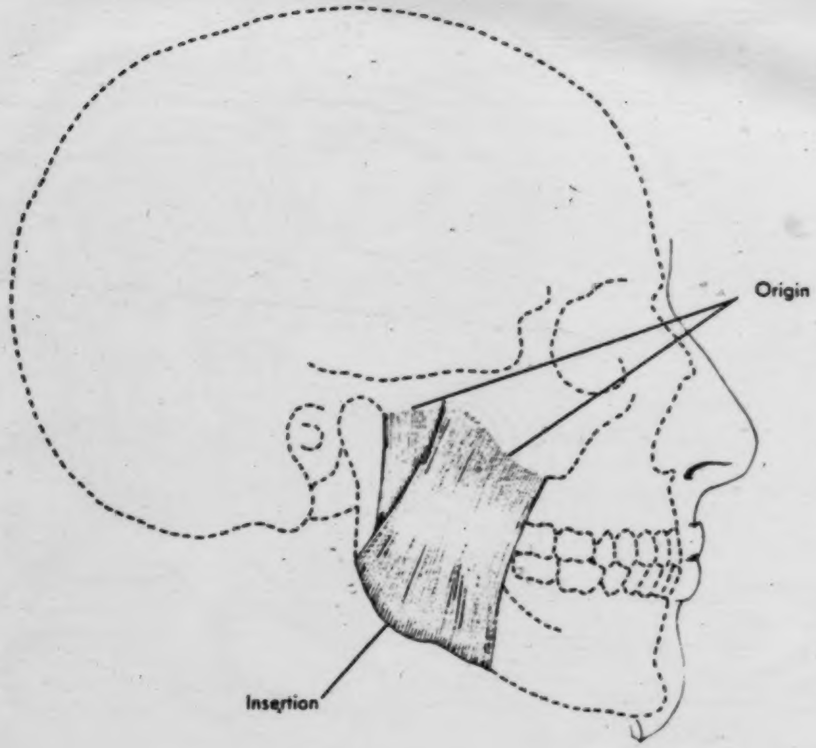
Lateral view of the skull (Anderson, 1978, p. 7-7).


Temporomandibular ligament
Capsule

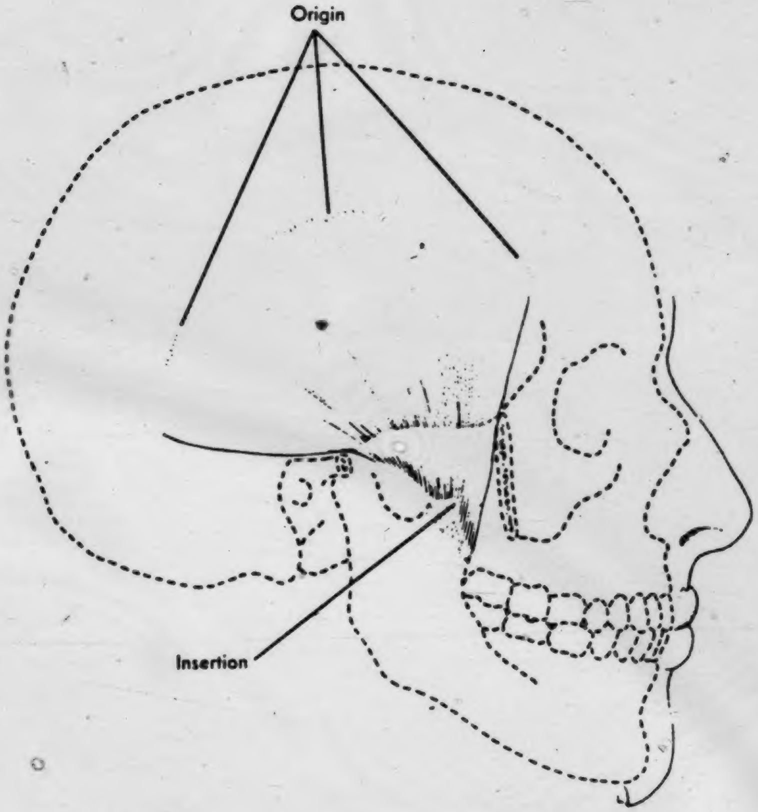
Capsule surrounding entire TMJ.
(Brand & Isselhard, 1982, p. 308).


Articular disc
Upper synovial cavity
Mandibular fossa
Articular eminence
Posterior tubercle
Lateral pterygoid muscle
Anterior attachment
Posterior attachment
Lower synovial cavity
Condyle

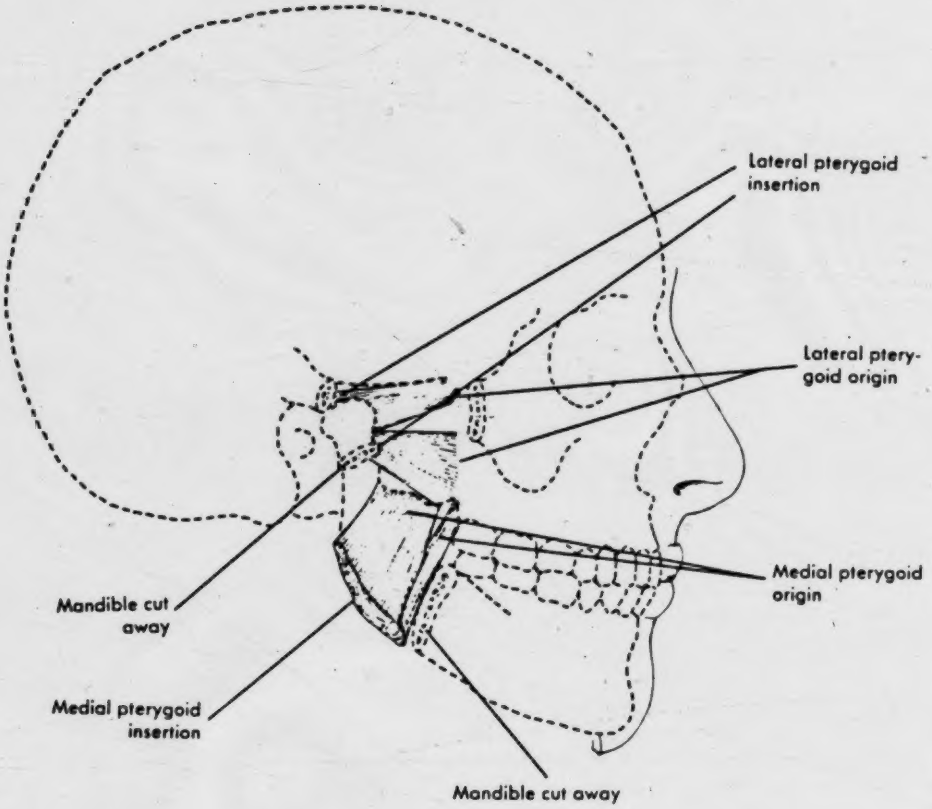
Longitudinal section through TMJ. Note disc and its anterior and posterior attachments. (Brand & Isselhard, 1982, p. 308).


Origin
Insertion

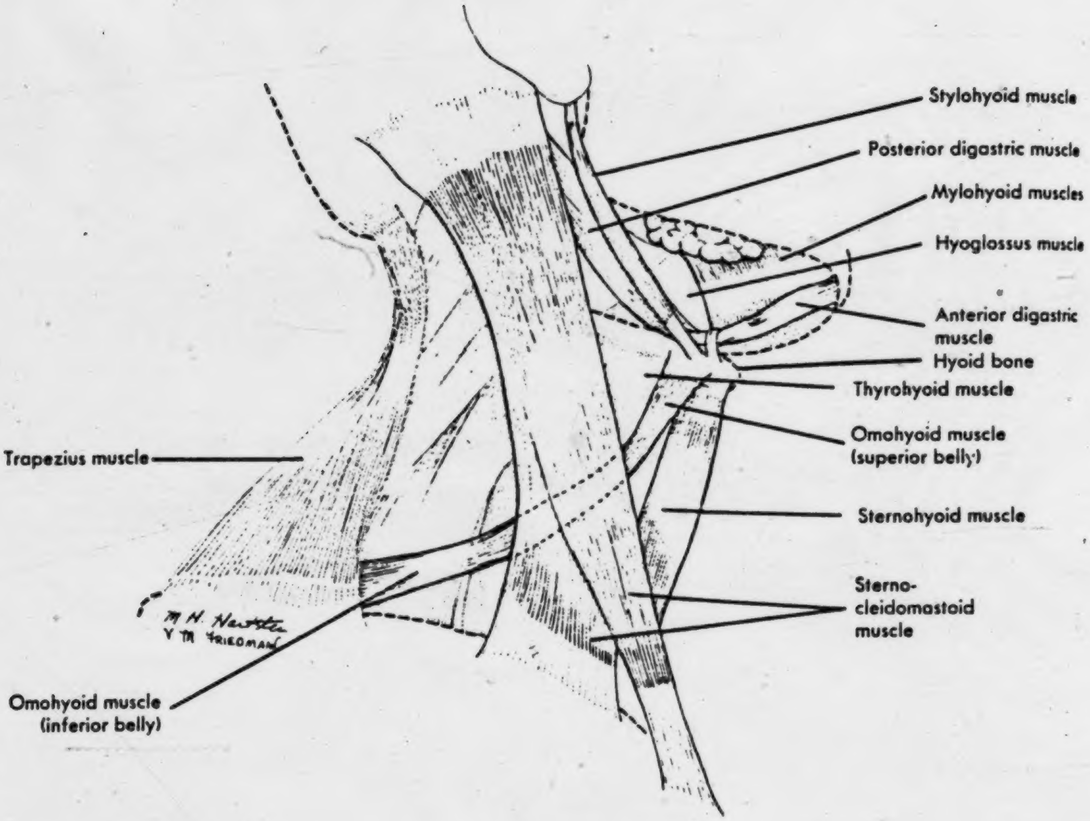
Lateral view of skull showing origin of masseter muscle from zygomatic arch. (Brand & Isselhard, 1982, p. 311).


Origin
Insertion

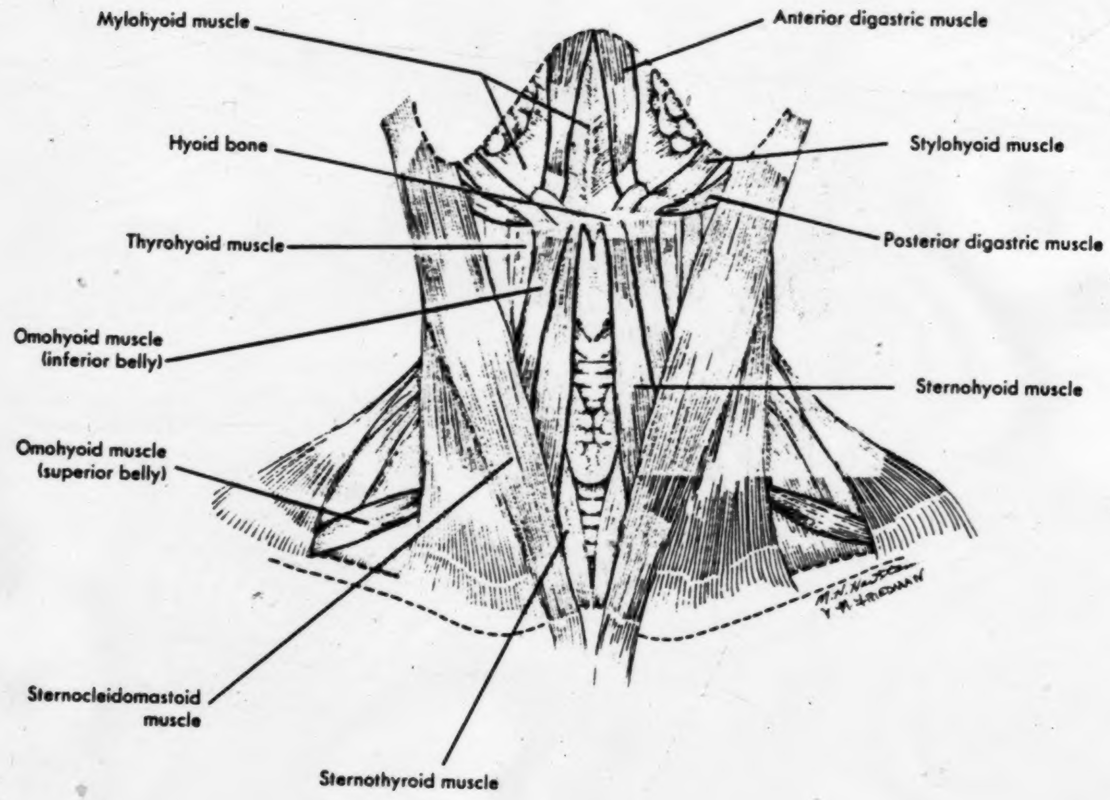
Temporalis muscle (Brand & Isselhard, 1982, p. 312).


Lateral pterygoid insertion
Lateral pterygoid origin
Medial pterygoid origin
Mandible cut away
Medial pterygoid insertion
Mandible cut away

Lateral view of the skull showing the lateral and medial pterygoid muscles. (Brand & Isselhard, 1982, p.313).


Stylohyoid muscle
Posterior digastric muscle
Mylohyoid muscles
Hyoglossus muscle
Anterior digastric muscle
Hyoid bone
Thyrohyoid muscle
Omohyoid muscle (superior belly)
Sternohyoid muscle
Sterno-cleidomastoid muscle
Trapezius muscle
Omohyoid muscle (inferior belly)
M.H. Netter

Lateral view of neck muscles.
(Brand & Isselhard, 1982, p. 314).


Mylohyoid muscle
Anterior digastric muscle
Hyoid bone
Stylohyoid muscle
Thyrohyoid muscle
Posterior digastric muscle
Omohyoid muscle (inferior belly)
Sternohyoid muscle
Omohyoid muscle (superior belly)
Sternocleidomastoid muscle
Sternothyroid muscle
M.N. NAJJAR
Y.M. FRIEDMAN

Anterior and inferior view of neck muscles.
(Brand & Isselhard, 1982, p. 315).

Appendix C

Questionnaire

1. Do you clench or grind your teeth during the day?
2. Have you been made aware of clenching or grinding your teeth during the night?
3. Do you have chronic headaches, or neck and shoulder pains?
4. Do you frequently have gastro-intestinal disturbances?
5. Do you ever wake up with an awareness of, or about, your teeth or jaw like you had them clenched in your sleep?
6. Do you have any awareness of the muscles of your neck or shoulders?
7. Do you have a tight or stiff neck?
8. Do you now, or have you ever had, pain in your jaw joint of the sides of your face (in and about the ears)?
9. Do you have a clicking jaw joint or have you ever experienced an inability to move your jaw or open your mouth widely?
10. Which side of your mouth do you chew on?
11. Do you tend to breathe mostly through your nose?
12. Are you aware of persistent ringing in your ears?
13. Have you ever experienced pain or burning sensations in your:
    a. neck
    b. shoulders

- c. back
- d. hips

14. Have you ever had treatment for problems of your:
    - a. neck
    - b. shoulders
    - c. back
    - d. hips
15. Have you ever been told you have:
    - a. scoliosis
    - b. lordosis (swayback)
16. Have you ever been told you have a leg shortening on one side?
17. Have you ever suspected a leg shortening on one side?

Appendix D

Experimental Procedure

The experimental component of the present study consisted of a number of stressors and recovery periods over which repeated measures of EMG activity were obtained. The outline of the experimental procedure is as follows:

1. 15 minute introduction and hookup
2. 8 minute resting baseline on four channels:
    a. left temporalis
    b. left masseter
    c. right temporalis
    d. right masseter
3. 2 minute serial subtraction
4. 2 minute recovery
5. 1 minute physical exertion (legs extended)
6. 1 minute recovery
7. 3 minute recorded soundtrack
8. 3 minute recovery
9. 3 minute cracker dissolving
10. 3 minute cracker mastication and recovery
11. 10 minute cleanup and debriefing

At the outset of the experiment, subjects were given a brief introduction to the nature and purpose of the study after which the EMG electrodes were attached. The sequence of the experiment and the particular stressors were not disclosed ahead of time. Subjects were led into the eight minute baseline with the following verbal directions:

> We will be starting the experiment shortly and I would ask you to sit quietly with your arms in a comfortable position, and both feet flat on the floor (a foot rest was provided for those subjects who could not reach the floor). I would like you to maintain this position with your eyes closed for the duration of the experiment, and please refrain from any extraneous movement. Unless you have any questions, I will begin the first recording period now, it will last for eight minutes.

After completion of the baseline period, the serial subtraction stressor was introduced:

> Now, I want you to take the number 1000, subtract 7 and get an answer. Subtract 7 again, get a new answer, and so on. Do this as fast as you can, silently, and without making any mistakes. I will ask you for your answer when the time is up. Do you understand the task? You may start now.

After the serial subtraction exercise, the recovery period was initiated: *Stop! The time is up. How far did you get? Now I just want you to relax, forget about the subtraction and return to sitting quietly.* The physical exertion stressor was introduced next: *Now I would like you to extend your legs straight out in front of you and sqeeze them together as hard as you can. Continue doing this until I tell you to stop. This will last for one minute.*

At the end of the sixty second recording period: "Stop! Now just return to relaxing with your feet flat on the floor." The recovery lasted one minute after which the soundtrack was presented:

> I am now going to place some headphones over your ears and play some music which I'd like you to listen to as you relax. The music is on a Sony Walkman, and the volume is set at four, it should not be too loud.

Once the headphones were properly positioned and adjusted, the music was started and EMG recordings were made for a three minute period. Following the music, the tape was stopped and the subjects were allowed to continue relaxing for a second three minute recovery period before the headphones were removed: *I will turn off the tape now, just continue relaxing.*

Once this recovery period had been completed, the headphones were removed and the subjects were given a small cracker with these instructions: *I would like you to take this cracker, place it on your tongue and let it dissolve without chewing it.* When the three minute time period had expired the subjects were offered a glass of water to wash down the cracker and then led into the final segment of the experiment: *Here is another cracker. Please take it, and when I give you the signal, put it in your mouth and eat it normally. When you have finished, rinse it down with some more water and then return to relaxing.* When this last time period had expired, subjects were told the experiment was over, and offered a brief synopsis of their performance as the electrodes were detached.

Appendix E

Summary of two-way analysis of variance N=48
Baseline, Serial subtraction

Source	*SS*	*df*	*MS*	*F*	*p*
Between subjects	2011.637	47			
A	1.148	1	1.148	.026	.871
Subjects within groups	2010.488	46	43.706		
Within subjects	812.617	48			
B	214.143	1	214.143	16.758	.00017
AB	10.658	1	10.658	.843	.36587
B X subjects within groups	587.824	46	12.779		

Summary of two-way analysis of variance N=48
Baseline, Serial subtraction recovery

Source	*SS*	*df*	*MS*	*F*	*p*
Between subjects	1831.102	47			
A	23.619	1	23.619	.601	.44213
Subjects within groups	1807.480	46	39.293		
Within subjects	288.145	48			
B	41.438	1	41.438	7.735	.00782
AB	.287	1	.287	.054	.81795
B X subjects within groups	246.418	46	5.357		

Summary of two-way analysis of variance N=48
Baseline, Physical exertion

Source	*SS*	*df*	*MS*	*F*	*p*
Between subjects	4827.754	47			
A	151.377	1	151.377	1.489	.22858
Subjects within groups	4676.379	46	101.660		
Within subjects	2247.863	48			
B	271.389	1	271.389	6.526	.01401
AB	63.492	1	63.492	1.527	.22288
B X subjects within groups	1912.988	46	41.587		

B

Summary of two-way analysis of variance N=48
Baseline, Physical exertion recovery

Source	*SS*	*df*	*MS*	*F*	*p*
Between subjects	1767.746	47			
A	37.313	1	37.313	.992	.32448
Subjects within groups	1730.434	46	37.618		
Within subjects	182.793	48			
B	13.471	1	13.471	3.729	.05965
AB	3.158	1	3.158	.874	.35466
B X subjects within groups	166.172	46	3.612		

Summary of two-way analysis of variance N=48
Baseline, Soundtrack

Source	*SS*	*df*	*MS*	*F*	*p*
Between subjects	1268.656	47			
A	31.658	1	31.658	1.177	.28357
Subjects within groups	1237.000	46	26.891		
Within subjects	144.148	48			
B	24.012	1	24.012	9.324	.00375
AB	1.676	1	1.676	.651	.42401
B X subjects within groups	118.465	46	2.575		

Summary of two-way analysis of variance N=48
Baseline, Soundtrack recovery

Source	*SS*	*df*	*MS*	*F*	*p*
Between subjects	1651.715	47			
A	60.229	1	60.229	1.741	.19356
Subjects within groups	1591.488	46	34.598		
Within subjects	166.977	48			
B	1.365	1	1.365	.408	.52608
AB	11.742	1	11.742	3.510	.06735
B X subjects within groups	153.876	46	3.345		

Summary of two-way analysis of variance N=48
Baseline, Cracker dissolving

Source	SS	df	MS	F	p
Between subjects	2064.293	47			
A	131.877	1	131.877	3.139	.08305
Subjects within groups	1932.414	46	42.009		
Within subjects	822.645	48			
B	227.168	1	227.168	19.196	.00007
AB	51.117	1	51.117	4.320	.04329
B X subjects within groups	544.363	46	11.834		